ANNALS OF SURGERY
VOL. 116 SEPTEMBER, 1942 No. 3

SYMPOSIUM ON INTESTINAL ATRESIA

INTESTINAL OBSTRUCTION IN THE NEWBORN

J. W. DUCKETT, M.D.

DALLAS, TEXAS

FROM THE DALLAS MEDICAL AND SURGICAL CLINIC, BAYLOR UNIVERSITY HOSPITAL, AND BRADFORD MEMORIAL HOSPITAL FOR BABIES, DALLAS, TEXAS

SURGICAL TREATMENT of congenital obstruction of the intestine in the newborn is at last beginning to produce encouraging results. Thirty years ago, not a single successful operation had been announced, and most cases had been described only as autopsy findings. More recently, a small but gradually increasing number of recoveries has been reported. To Dr. William E. Ladd, Dean of pediatric surgery in this country, must go most of the credit for the achievements of surgery in this new field. Not only has Doctor Ladd called attention to the prevalence of this condition and stimulated efforts at making early diagnoses, but he has made valuable contributions to the operative technic and developed an original operation for relief of the extrinsic type of obstruction. The subject is presented in detail by Doctors Ladd and Gross in their recent volume entitled "Abdominal Surgery of Infancy and Childhood." Their discussion of congenital obstruction is based on a series of 118 cases treated in the Boston Children's Hospital, with 44 recoveries. The extent of this experience, however, is unique; and a search of the literature on the subject shows only reports of single cases or small groups. We feel justified, therefore, in reporting our own group of six cases, five of which were operated upon, with three recoveries.

Congenital obstruction of the intestine should be suspected whenever a newborn baby begins vomiting persistently soon after birth. The vomitus is usually bile-stained. It quickly becomes projectile, and everything taken by mouth is promptly lost if the obstruction is complete or almost so. The site of the obstruction cannot be predicted by the time-interval between birth and onset of vomiting, since the entire intestinal tract above the obstruction is already distended at birth by digestive juices and swallowed amniotic fluid. When air and fluids are swallowed in the first feeding efforts, distention is further increased and vomiting results. When obstruction is incomplete or intermittent, vomiting may begin later and may be less persistent, making diagnosis more difficult.

Anatomically considered, congenital obstruction may be intrinsic or extrinsic. The intrinsic obstruction is more frequent and may occur at any

location. It results from failure of reestablishment of the bowel lumen during the early weeks of fetal development. Proliferation of epithelium obliterates the lumen of the primitive gut for a time, but normally the lumen reappears after confluent vacuolization of the central mass. If this process is incomplete, one or more septa may remain to block the lumen or sections of the intestine may be represented by solid fibrous cords or threads. The condition is known as atresia when the bowel lumen is completely obstructed or when there is loss of continuity and as stenosis when a small but ineffective opening is present. In either group, the clinical picture is essentially the same, though in stenosis the abnormalities are more likely to be amenable to surgical treatment.

Extrinsic obstruction usually is a result of incomplete rotation of the colon associated with abnormally placed folds or bands of peritoneum which most frequently impinge upon the lower half of the duodenum.

Diagnosis of congenital atresia or stenosis can often be made on the history alone. In the absence of intracranial birth injury or unusual infection, bile-stained projectile vomiting beginning soon after birth and persisting, almost invariably means obstruction of the intestine. Hypertrophic pyloric stenosis can be differentiated by the later onset of vomiting and absence of bile from the vomitus.

Positive diagnostic aid may often be obtained from roentgenologic studies. Plain films of the abdomen are usually adequate, outlining the gas-filled obstructed stomach and intestine with striking clearness. Barium may be given if necessary but is to be avoided if possible because of danger of vomiting and aspiration into the lungs, and because masses of barium, difficult to remove by lavage, may interfere with operative procedures.

Survival of infants with complete obstruction depends on early diagnosis, and immediate repair of the defect. Without surgery, death invariably occurs within a week or ten days when obstruction is complete, and high degrees of partial obstruction are very poorly tolerated. Close coordination of medical and surgical care is an essential factor in survival. There is often jaundice with consequent bleeding tendency, which must be combated with vitamin K. Treatment of metabolic disturbances, especially dehydration, require preoperative administration of fluids and blood transfusion. Ether anesthesia is preferable because of relaxation needed for adequate exploration. An incision of considerable length is advisable to facilitate the extensive operative procedure often necessary. In cases of intrinsic obstruction, the best chance of a favorable outcome is offered by the performance of a primary anastomosis about the site or sites of obstruction. Two-stage procedures are poorly tolerated. In extrinsic obstruction, Ladd has demonstrated that the most satisfactory results are to be obtained by releasing the peritoneal band lying across the duodenum and reducing the volvulus of the small intestine often associated with this lesion. Certain important details of surgical procedure are essential to success in operations on these tiny patients. Body heat must be preserved and any appreciable change in temperature avoided. The delicate

tissues must be handled with extreme gentleness and complete hemostasis obtained. Postoperative care should include transfusion with whole blood or plasma, and care to avoid vomiting and distention by use of lavage and suction.

The following case reports include five intrinsic obstructions and one of the extrinsic type. Of the intrinsic group, one, which had a jejunal atresia, died before operation could be attempted. Two others with jejunal

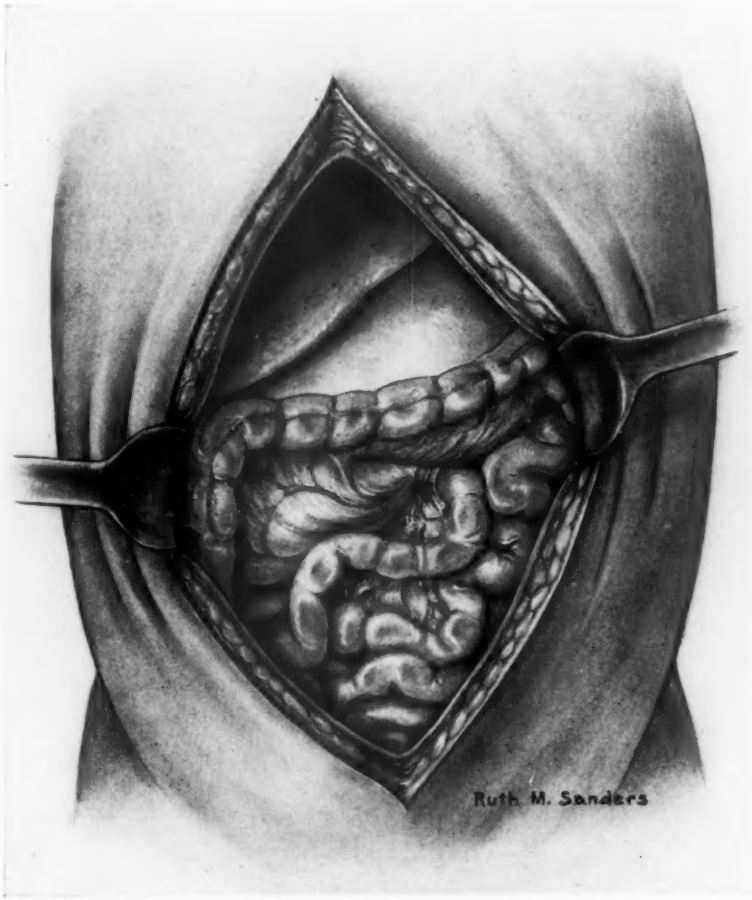


FIG. 1.—Case 1: Stenosis of duodenum near duodenojejunal junction. Findings in Case 6 were identical.

atresia were operated upon unsuccessfully, one dying 17 days after operation and the other 12 days postoperative. Both of these children had defects of the intestinal tract which were so extensive that physiologic function could not be restored, although satisfactory anastomoses were provided. Both of the cases of duodenal stenosis survived operation and are apparently developing normally, as is also the single case of extrinsic obstruction, upon which the Ladd operation was performed.

CASE REPORTS

Case 1.—Baby E., a white female, was born at Baylor Hospital, 2:00 A.M. July 6, 1938. Full term. Normal delivery. Birth weight: Six pounds, four ounces. Mother had hydramnios. Nursed with some difficulty but did not vomit for 48 hours after birth. One small stool of meconium passed. Vomiting became frequent and was bile-stained.

Physical Findings: Normal except for moderate abdominal distention and occasional visible peristaltic waves from left to right in the upper abdomen.

On July 8, 1938, a plain roentgenogram showed stomach and duodenum moderately distended with gas. Next day, barium was given and definite evidence of obstruction of duodenum obtained. Weight: Five pounds, five ounces. *Clinical Diagnosis:* Congenital obstruction of duodenum.

Operation.—July 10, 1938: Under ether anesthesia, a right upper rectus incision was made, through which the upper abdomen was explored. The stomach and duodenum were found to be greatly dilated and thick-walled. It was apparent that the obstruction of the lumen was due to an intrinsic defect, such as a persistent diaphragm. The jejunum and large intestine were normal in appearance wherever seen. The liver was normal (Fig. 1).

A short-circuiting operation was performed, anastomosing the side of the third portion of the duodenum with the side of the proximal end of the jejunum. A rent was made in the mesocolon, through which the wall of the duodenum was brought and attached by stay-sutures of No. 0000 chromic catgut to the antimesenteric border of the jejunum as near as possible to its origin. An anastomosis, with a stoma about two centimeters long, was made, using two layers of No. 0000 catgut sutures on atraumatic needles. When the duodenum was opened, a catheter was passed upward into the stomach, meeting no obstruction, and was then passed downward to the point of obstruction at the base of the mesocolon. Wound closed with continuous fine catgut suture in the peritoneum, interrupted figure-of-eight A-silk sutures in the anterior rectus sheath, and interrupted A-silk sutures in the skin. *Postoperative Diagnosis:* Congenital stenosis of duodenum.

Postoperative Course: CO_2 and O_2 were administered 24 hours to combat cyanosis Transfusion, saline and glucose given parenterally. Water one ounce every hour was started after 12 hours, and most of it retained. On third day, dilute breast milk was started, and the baby vomited small amounts twice. After this, there was no vomiting; and she was fed from the breast, gaining weight steadily. Discharged with mother, July 26, 1938, 16 days after operation. Weight: Five pounds, 15 ounces. The wound healed firmly, although there was a delay of three weeks in the healing of the skin edges.

This child has been seen at frequent intervals since operation. Her development has been entirely normal in every way, and she has enjoyed excellent health.

Case 2.—Baby W., white male, was admitted June 1, 1941. Born 2:45 A.M. May 29, 1941. Twenty days premature. Birth weight: Six pounds, eight ounces. Had vomited everything taken since birth No bowel movements. On admission, weight five pounds, four ounces. Moderate dehydration. Slight jaundice. Lungs clear and fairly well expanded. Heart sounds clear. No murmurs. Ticktack rhythm. Abdomen slightly distended, soft. No masses palpable. No visible peristalsis. Urine normal. Hb. 64 per cent. R.B.C. 3,950,000. W.B.C. 8,000. P.M.N. 38. Prothrombin time five minutes. Kline negative. No stool passed. After gastric lavage, a thin mixture of barium was given. Fluoroscopy and roentgenograms showed esophagus and stomach to be greatly dilated and beneath these a large V-shaped shadow, probably representing duodenum and upper jejunum, also greatly dilated.

Course in Hospital: Fluids and glucose were given parenterally. Nothing by mouth was allowed. Transfusions were given. No stools were passed other than a few particles of greenish material, which contained no bile. Vitamin K was given, and

prothrombin time was lowered to one and one-half minutes. *Clinical Diagnosis:* Congenital atresia of the small intestine.

Operation.—June 3, 1941: Under light ether anesthesia, a right paramedian incision was made. Almost the entire abdominal cavity was occupied by the dilated stomach, continuous with an enormous loop of distended small intestine. The exact identity of this loop could not be determined, although it had the appearance of a greatly enlarged

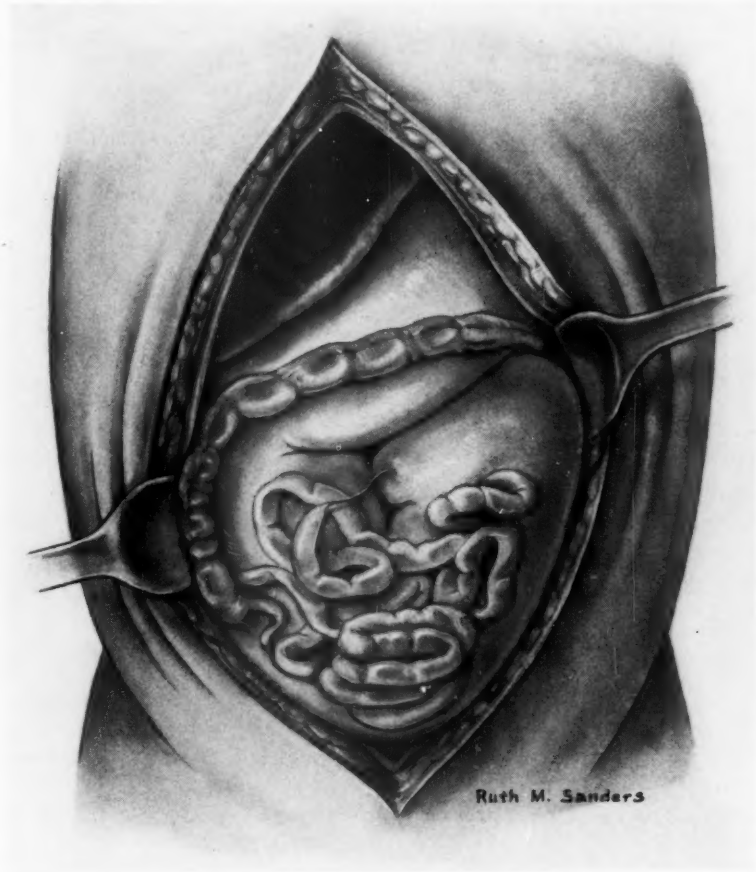


FIG. 2.—Case 2: Atresia of jejunum and abnormal attachment of ascending and transverse colon.

duodenum, extending downward in the right side of the abdomen, into the pelvic cavity, and up the left side under the diaphragm, then medially to end blindly in the usual location of the duodenojejunal junction. Here the intestine was replaced by a fine fibrous cord three centimeters in length, which joined the dilated portion and the beginning of a normal looking small intestine, completely collapsed. The cecum, appendix, and ascending colon lay loosely adherent to the anterior surface of the descending limb of the dilated loop. The large intestine was collapsed but normal in appearance as far as it could be traced to a point where it was concealed by the ascending portion of the dilated loop. The liver and gall bladder were normal in appearance (Fig. 2).

An anastomosis was made between the distal end of the dilated intestine and the collapsed jejunum near its proximal end. Two layers of No. 0000 chromic catgut were

used. The abdominal wound was closed with through-and-through silk sutures. *Postoperative Diagnosis:* Congenital atresia of the jejunum; abnormal attachment of ascending colon.

Postoperative Course: Given concentrated blood plasma intravenously immediately after operation. Condition was good for several days. Nothing was given by mouth for 48 hours, then water in small amounts was given but vomited immediately. Vomiting continued; no stools were passed. Normal saline solution was injected into the lower bowel and promptly expelled. Prostigmine caused audible peristaltic activity for a few minutes but no stool resulted. Temperature remained constantly subnormal. Upper respiratory infection appeared. Death occurred at 12:45 P.M. June 20, 1941. Autopsy permission was refused.

Case 3.—Baby N., a white female, was admitted, June 11, 1941, 12 hours after birth. Birth normal. Full term. Weight: Four pounds, six ounces. Mother had polyhydramnios. Baby began vomiting immediately after birth and this continued at frequent intervals until death. No stools were passed. The baby's body had felt cold and on admission temperature was 97.8°, falling later to 96.4°. Aside from the small size and low temperature, there were no abnormal physical findings.

Urine: Albumin—a trace, rare W.B.C., and casts. Hb. 20.8 Gm. R.B.C. 5,800,000, W.B.C. 9,600. P.M.N. 39. Prothrombin time six minutes. Kline negative.

Course in Hospital: Small amounts of greenish fluid were vomited frequently, although the stomach was lavaged repeatedly and nothing given by mouth for 24 hours. A weak milk formula was then given and promptly vomited. Transfusions and intravenous saline and glucose were given. On the third day, a few particles of greenish material were passed from the bowel. On June 15, 1941, four days after birth, the child died, with a sudden rise of temperature to 104°. The probable diagnosis was "birth injury." No surgical consultation had been requested.

Autopsy Report.—Anatomic Diagnosis:

1. Congenital abnormalities of gastro-intestinal tract:
   a. Lack of continuity of duodenum and jejunum.
   b. Marked dilatation and distention of duodenum, stomach, and esophagus.
   c. Partial atresia of jejunum.
   d. Small ascending, transverse, and descending colon.
   e. Absent omentum.
2. Prematurity.
3. Fetal atelectasis.
4. Small right ventricle.
5. Patent ductus arteriosus.
6. Mild icterus.
7. Uric acid infarcts of kidneys.

"On making the usual midline incision, there is a very thin layer of granular fat present in the abdominal wall. The peritoneal surfaces are smooth and glistening throughout, and there is no excess of fluid. A markedly dilated loop of intestine presents itself, and this is later identified as markedly dilated duodenum. The stomach is properly rotated in its correct location; it is markedly distended with gas. The constriction of the pylorus is readily identified. From this point onward, the duodenum is markedly distended, and in this manner has displaced the remainder of the gastro-intestinal tract downward. The first portion of the duodenum is attached to the visceral surface of the liver by means of peritoneum; from this region the duodenum pursues its usual U-shaped course to the left, being related to the right kidney and suprarenal gland. It ends as a blind pouch without any connections to the jejunum. No atretic connection can be made out. The jejunum commences as a blind pouch of fair size, and this is connected to the remainder of the jejunum by means of a fibrous cord; this pouch is entirely separated from the duodenum, and the two lumina are not continuous. The remainder of the jejunum and ileum are of the usual appearance; the appendix is in the

right lower quadrant. The entire colon appears to be much smaller than usual; the transverse portion is enveloped by the visceral peritoneum of the duodenum and stomach. There is no omentum present (Fig. 3).

"On opening the chest, the lungs fill their respective spaces; there are no adhesions. The pleural surfaces are smooth and glistening, and there is no excess fluid. The thymus gland is not enlarged, is bilobed and composed of soft, grayish tissue.

"On opening into the pericardial sac, no excess fluid is present; and the surfaces are smooth and glistening. The superior and inferior venae cavae are markedly distended

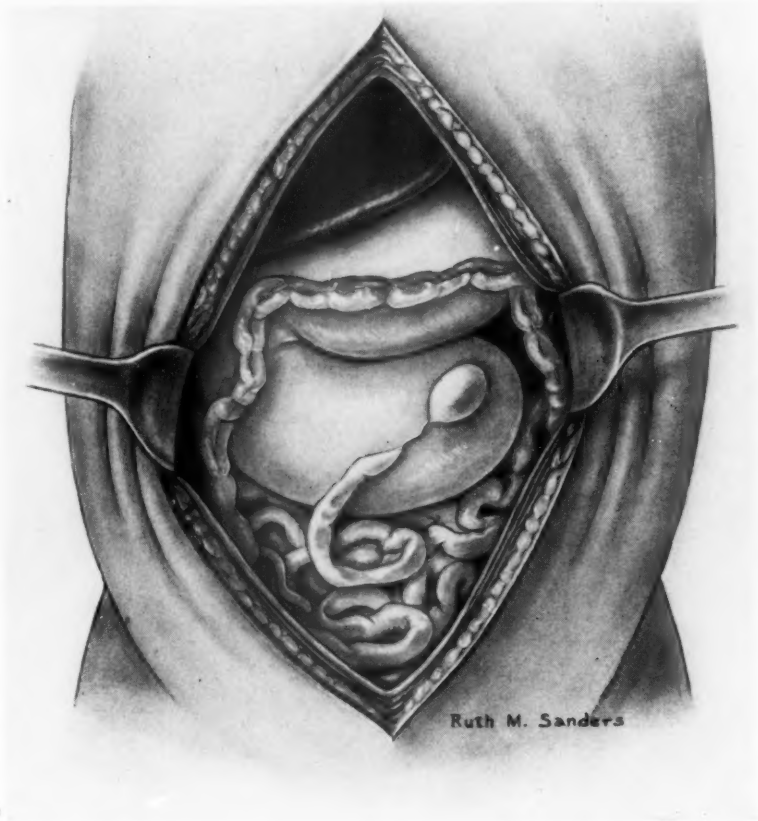


FIG. 3.—Case 3: Atresia and aplasia of jejunum.

with blood. The heart is of normal size, and there are no abnormalities about the aortic arch or its main branches.

"Both lungs float in water. The right lung is trilobed and of dark reddish-purple color; it fails to crepitate and is flabby. The larger bronchi are of the usual caliber and are not obstructed. On section, the cut-surface is dark red in color; and a minimal amount of air can be expressed. The left lung is bilobed and resembles its fellow in all respects.

"The left auricle of the heart is empty, and the foramen ovale is functionally closed. The chamber of the right ventricle is much smaller than usual; there are no abnormalities about the tricuspid valve. The cusps of the pulmonary valve are intact. There are no changes noted about the mitral or aortic valves. The myocardium is deep red in color and of good tone."

Case 4.—Baby M., a colored female, was admitted June 28, 1941. Born 1:00 A.M. June 27, 1941. Full term. Normal delivery. Had received nothing by mouth except water in small amounts, and had vomited greenish fluid persistently. There had been no bowel movement. Weight: Five pounds, eight ounces. Except for slight dehydration, physical examination was normal. The abdomen was soft, and no masses were felt.

Urine contained albumin, and a rare pus cell. Hb. 11 Gm. R.B.C. 3,000,000. W.B.C. 8,000. P.M.N. 98. Kline negative.

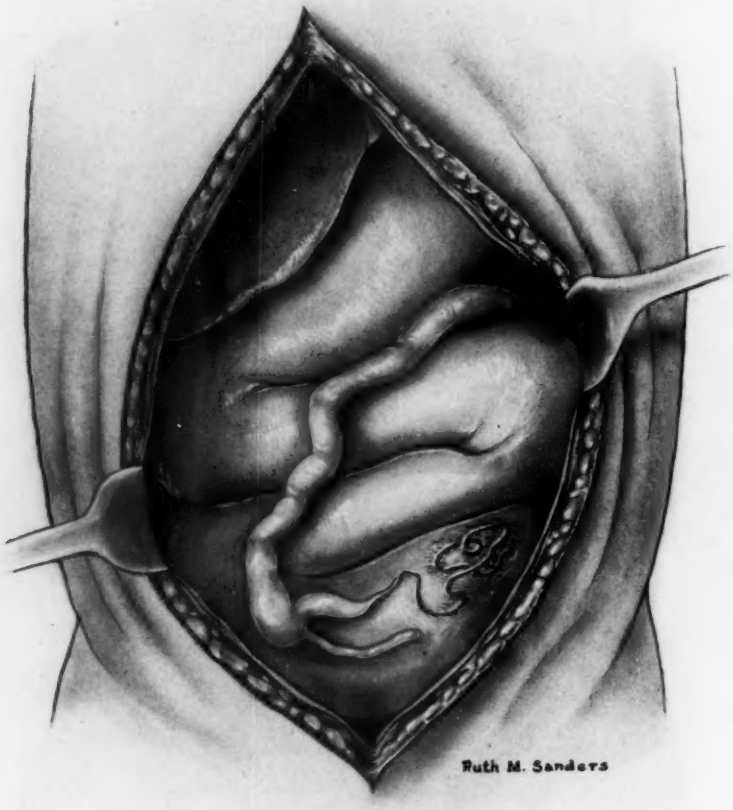


FIG. 4.—Case 4: Atresia of jejunum and aplasia of ileum. Abnormal attachment of ascending and transverse colon.

After oral administrations of barium, roentgenograms showed a great dilatation of the stomach and upper part of the small intestine, which were partly filled with barium. Below this, was more gas-filled intestine. Barium enema showed a small but normal appearing colon.

Course in Hospital: Nothing was given by mouth, but vomiting continued. Saline and glucose were given parenterally as well as blood transfusion. Vitamin K was given in preparation for operation. *Preoperative Diagnosis:* Congenital obstruction of small intestine.

Operation.—June 30, 1941: Under light ether anesthesia, a right paramedian incision, five centimeters long, was made. The abdominal cavity was almost entirely filled by a

large sac-like dilatation of stomach and upper part of the small intestine. The first impression was that the main part of the cystic mass represented a huge dilated and hypertrophied duodenum, but closer inspection showed transverse folds dividing the mass into a series of three loops, closely pressed together and adherent. This dilated portion of the small intestine ended blindly in the left lower quadrant of the abdomen, and the remaining portion of the small intestine was represented by a small, twisted cord-like structure intimately adherent to the anterior surface of the terminal dilated loop. The cord connected with the proximal end of a very short terminal ileum, six centimeters in length, which was of normal diameter and possessed a lumen. The cecum, appendix, and ascending colon were small and collapsed, and lay on the anterior surfaces of the dilated loops of small intestine, running upward to the right of the midline to disappear under the greater curvature of the stomach. The wall of the dilated intestine was thick, slightly edematous, and hyperemic (Fig. 4).

An anastomosis was made between the side of the short terminal ileum and the blind end of the dilated intestine. Two layers of No. 0000 chromic catgut were used. The abdominal wound was closed with through-and-through C-silk sutures. *Postoperative Diagnosis:* Congenital atresia of jejunum; aplastic ileum.

Postoperative Course: Operation completed with no apparent shock. Concentrated blood plasma given intravenously. Stomach was lavaged repeatedly, and nothing given by mouth for three days. Vomiting continued, but less frequently. Saline solution was injected into the rectum to stimulate bowel activity but no stool was passed until the seventh postoperative day, when a few shreds of greenish material were expelled. Similar scanty stools were passed several times thereafter. After the third day, weak glucose solution and, later, an evaporated milk formula were given by mouth, but vomiting continued. Transfusions and parenteral fluids were given daily. Jaundice developed, followed by upper respiratory infection. Death occurred, July 12, 1941, on the twelfth postoperative day.

Autopsy Findings.—Anatomic Diagnosis:

1. Congenital atresia and aplasia of ileum.
2. Marked dilatation of jejunum and duodenum.
3. Surgical anastomosis of terminal ileum and jejunum.
4. Marked jaundice.
5. Chronic fibrous adhesions in peritoneal cavity.
6. Slight diminution in rotation of colon.
7. Patent ductus arteriosus.
8. Patent foramen ovale.
9. Confluent bronchopneumonia of both lungs.

"The fat in the midline is deep yellow in color. On opening into the peritoneal cavity, the loops of the intestine are markedly adherent by fibrous tissue. There are several markedly distended loops of small intestine. The stomach and duodenum are in their usual locations but are markedly distended, with average diameter of 3.5 cm. The jejunum is also markedly distended and makes three loops, which, together with the stomach and duodenum, occupy at least 75 per cent of the peritoneal cavity. On tracing the intestinal loops, the jejunum ends in a blind dilated sac about 12 inches from its commencement. The remainder of the small intestine is represented by a narrow fibrous cord coiled on the anterior surface of the dilated jejunum. The terminal six centimeters of the ileum has a very small lumen of about 0.5 cm. in diameter. This portion of the bowel has been anastomosed to the dilated jejunum. The stoma of this anastomosis is open but the distal segments contain no air and only a small amount of semisolid grayish-yellow material. The stoma of the anastomosis measures four millimeters in diameter, and the margins are slightly edematous. The cecum and the ascending colon are flattened and are situated medial to the normal position so that they lie in approximately the midline. The remaining portions of the large intestine are also

flattened but the lumen is patent as in the rectum. The remaining viscera are in their usual locations.

"The liver border is 2.5 cm. below the costal margin. The diaphragm on the right is at the level of the 4th rib and on the left at the 4th interspace. The thymus is of the expected size.

"Both pleural cavities are free from excess fluid, and their surfaces are smooth and glistening. The pericardial surfaces are smooth and glistening. The heart is of normal size and the ductus arteriosus is open. The endocardial surfaces are everywhere smooth and glistening. The foramen ovale is open but closed by a flap. The various valves are firm, and the leaflets are normal. The myocardium is dark reddish-gray.

"The lungs are similar in general appearance, both being heavy, dark reddish-gray and airless. On section, the lung tissue is bluish and contains many confluent, firm consolidated areas. A moderate amount of frothy, yellowish fluid can be expressed from the upper lobes."

Case 5.—Baby B., a white female, was born at Baylor Hospital October 10, 1941. Full term. Normal delivery. Weight: Seven pounds, four ounces. Vomiting first occurred when baby was three days old and was intermittent thereafter. Vomitus was bile-stained several times. Visible peristalsis was recorded at times, waves passing from left to right in the upper abdomen. On October 22, 1941, barium was given by mouth, and some delay in emptying of the stomach was noted; but no organic obstruction could be demonstrated. On October 26, 1941, the baby was discharged, having shown some improvement on thickened feedings and atropine. Vomiting had at times been projectile. Stools were normal in appearance and amount.

After leaving the hospital, symptoms became promptly worse. Projectile vomiting after almost every feeding began. A small tumor was thought to have been felt in the region of the pylorus. Weight dropped to six pounds, two ounces.

On October 31, 1941, the baby was readmitted to the hospital. *Clinical Diagnosis:* Hypertrophic pyloric stenosis. Saline and glucose given by hypodermocylsis.

Operation.—November 1, 1941: Under light ether anesthesia, a short right upper rectus incision was made and later lengthened. The stomach and duodenum were moderately dilated, and, at the pyloric ring, the muscle fibers were moderately thickened, but there was no definite pyloric tumor; small intestine, dilated. Below the duodenojejunal junction, the small intestine was collapsed and cyanotic. The cecum was found in the left upper quadrant of the abdomen, where it was held by a thin fold of peritoneum, which extended across the duodenojejunal ligament to the iliac fossa on the right. The entire small intestine was found to be rotated at its mesenteric attachment through 360° in a counter-clockwise fashion. The sigmoid colon was extremely long, extending into both upper quadrants. In addition to the circulatory obstruction of the small intestine, there was obstruction of the lower portion of the duodenum just above the duodenojejunal junction by the abnormal peritoneal fold, to which the cecum was attached. There was no attachment of the mesentery of the small intestine except at the point of entrance of the superior mesenteric artery (Fig. 5).

The stomach was first examined, and the thickening at the pyloric ring incised longitudinally through an incision about one centimeter long. The muscle fibers were separated and the mucosa allowed to bulge through the incision to the level of the serosa. The abnormal peritoneal fold was sectioned, allowing the volvulus to be reduced by clockwise rotation through 360°; and the cecum was then replaced in the right lower quadrant, but no attempt was made to attach it. After reduction, the color of the small intestine became normal and the intestine rapidly filled with gas. The wound was closed in layers without drains, using fine cotton No. 100 throughout. *Postoperative Diagnosis:* Extrinsic obstruction of the duodenum; incomplete rotation of the cecum; volvulus of the small intestine; early hypertrophic pyloric stenosis.

Postoperative Course: Saline and glucose solution given by hypodermoclysis. Nothing was given by mouth for 24 hours; then water in small amounts was started

and retained. Thirty-six hours after operation, a formula of equal parts evaporated milk and water was given in half ounce amounts and mostly retained. The amount of the formula was increased gradually to three ounces every three hours, but, on the fifth postoperative day, vomiting of about half of each feeding began. Thereafter, the adjustment of feedings continued to present a problem; but, when discharged, November

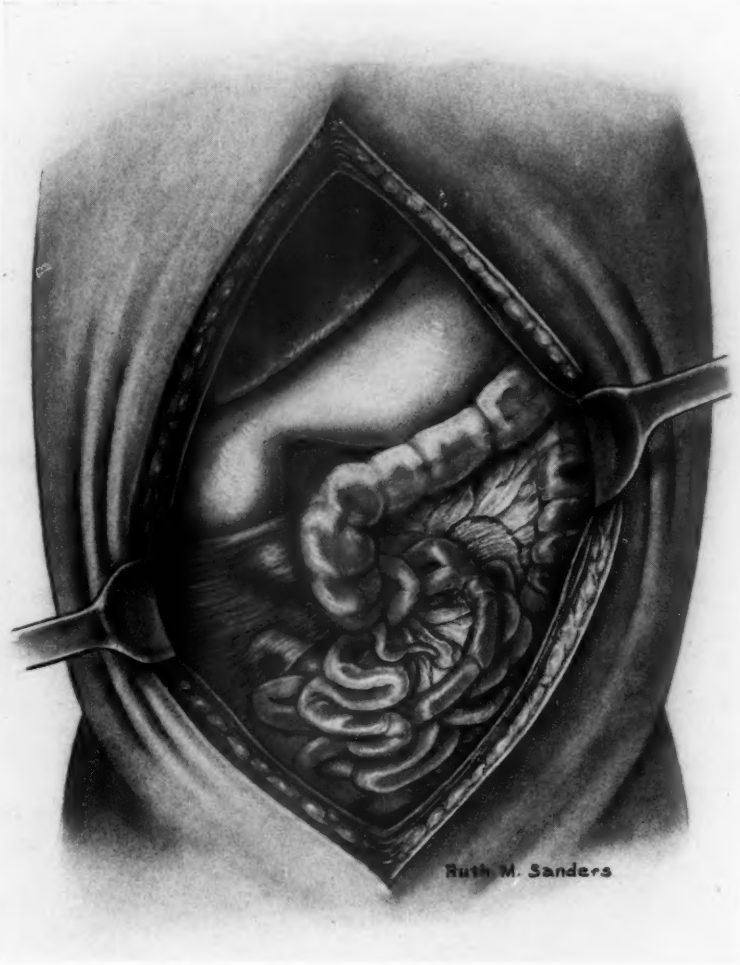


FIG. 5.—Case 5: Malrotation of colon; duodenal obstruction by band; volvulus of small intestine.

11, 1941, vomiting was occurring only once or twice a day, and there was a steady gain in weight. This course continued for the next three weeks; then vomiting ceased and has not recurred. Normal development has continued.

Case 6.—Baby D. a white male, was born at Good Samaritan Hospital December 6, 1941. Full term pregnancy. Difficult delivery with high forceps, resulting in two cephalhematomata. When two days old, the baby had a convulsion and began vomiting. One small bowel movement of meconium was passed.

Admitted to Bradford Memorial Hospital, December 9, 1941, vomiting frequent small amounts of bile-stained fluid and retaining nothing taken by mouth. Barium was

given by mouth and roentgenograms showed a greatly dilated stomach and duodenum. No barium passed beyond the duodenum.

Weight: Five pounds, eight ounces. There was moderate dehydration and slight jaundice. A large cephalhematoma was present on each side of the head in the occipitoparietal regions. There was no cyanosis and no sign of increased intracranial pressure. The abdomen was slightly distended, and peristaltic waves passing from left to right across the upper abdomen were noted at times. Examination was otherwise normal. *Clinical Diagnosis:* Duodenal atresia. Saline and glucose solutions and a blood transfusion were given in preparation for operation.

Operation: December 11, 1941: Under light ether anesthesia, a right upper rectus incision was made. The stomach and duodenum were greatly dilated and hypertrophied, and the pyloric ring was identified with difficulty, since the pylorus and duodenum were uniformly dilated to a diameter of about five centimeters. The point of obstruction was at or near the duodenojejunal junction, and was intrinsic. The jejunum and the remaining portions of the small intestine were normal in appearance, but completely collapsed. Cecum, appendix, and ascending colon were also normal. The transverse colon was displaced upward and the transverse mesocolon stretched over the enlarged duodenum (Fig. 1).

The dilated stomach and duodenum were collapsed by catheter passed through the mouth and the location of the obstruction was identified. The entire small intestine was examined and found to be normal. An incision was made through the mesocolon at an avascular area, through which the lowermost part of the wall of the duodenum was drawn and attached by an anchoring suture to the proximal portion of the jejunum as close to the duodenojejunal junction as possible. A side-to-side anastomosis was made, using two layers of fine cotton sutures. The stoma was about 2.5 cm. in length. The wound was closed in layers, using fine cotton sutures.

Postoperative Course: Concentrated blood plasma given intravenously immediately after operation. Nothing was given by mouth for 24 hours; then water four cubic centimeters every hour, was started and increased gradually as no vomiting occurred. Forty-eight hours after operation, breast milk was given but part of each feeding was vomited. Vomitus was heavily bile-stained. A regimen of gastric lavage immediately preceding each feeding was begun and vomiting promptly stopped. Normal stools were passed daily after feedings began. Weight gain was slow but increased from five pounds two ounces, before operation to six pounds, thirteen ounces at discharge, January 10, 1942, 30 days after operation. Wound healing was delayed by small areas of subcutaneous suppuration, but, at discharge, there was a firmly healed scar. Development proceeded normally. On February 11, 1942, the baby weighed nine pounds, and was apparently in excellent health.

Discussion.—It is significant that the last five of these six cases were encountered during a period of six months, while the first was operated upon three years earlier. This may be coincidence, but it is more likely explained on the basis of an increased awareness of the occurrence of congenital obstruction in the newborn. It is encouraging to feel that more and more early diagnoses will be made, and that the list of successful operations will grow rapidly larger. We have no original contributions to make either to diagnostic methods or surgical procedure. We have been impressed, however, with the beneficial effect of concentrated blood plasma in the postoperative treatment of our recent cases. When plasma has been given intravenously immediately after operation, no signs of shock have occurred and the postoperative course in each case has been astonishingly smooth. Another technical detail of possible interest is the use of very fine cotton thread No. 100, as

the only suture material. Except for a minor skin infection in one case, we have encountered no difficulty in wound healing when fine cotton has been used throughout the operation. It seems particularly adapted for this type of surgery.

REFERENCES

[1] Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood. Philadelphia, W. B. Saunders Co., 1941.

[2] Ladd, W. E.: Congenital Duodenal Obstruction. Surgery, **1**, 878, 1937.

[3] Miller, E. M.: Bowel Obstruction in Newborn. ANNALS OF SURGERY, **110**, 587, October, 1939.

[4] Cohen, P.: Congenital Intestinal Obstruction. Am. Jour. Dis. of Child. **61**, 135, January, 1941.

[5] Glover, D. M., and Hamann, C. G.: Intestinal Obstruction in the Newborn Due to Congenital Anomalies. Ohio State Med. Jour., **36**, 833, August, 1940.

[6] Elman, Robert: Ladd's Operation for Cure of Incomplete Rotation and Volvulus of the Small Intestine Producing Duodenal Obstruction in Infancy. ANNALS OF SURGERY, **112**, 234, August, 1940.

[7] Donovan, Edward J.: Congenital Atresia of the Duodenum in the Newborn. ANNALS OF SURGERY, **103**, 455, March, 1936.

[8] Stetten, De Witt: Duodenojejunostomy for Congenital Intrinsic, Total Atresia at the Duodenojejunal Junction. ANNALS OF SURGERY, **111**, 583, April, 1940.

[9] Lee, Walter Estell: Discussion of Stetten.[8]

[10] Donovan, Edward J.: Discussion of Stetten.[8]

ATRESIA OF THE DUODENUM

CASE REPORT

ROBERT R. IMPINK, M.D., AND

GEORGE R. CLAMMER, M.D.

READING, PA.

THE NUMBER of reported cases of duodenal atresia is small, and the management of such cases fraught with difficulties and disappointments. When infants with this anomaly are encountered, therefore, it is important to record their history in the hope that the addition of some minor detail to the existing knowledge of the condition may aid in the diagnosis and treatment of future patients. We are, therefore, presenting the following case:

Case Report.—B. B., a male infant, 30 hours old, was admitted to St. Joseph's Hospital, March 7, 1941. His birth had been uncomplicated. Both parents were living and well, as were seven siblings. One sibling had died of pneumonia in his first year.

When only six hours old the infant vomited a small amount of dark brown fluid. Such vomiting recurred at varying intervals after the ingestion of fluids during the first 24 hours, and caused the physician in charge, Dr. Charles B. Bertolet, to send the child to the hospital. At the time of admission the patient's general condition was good. There was no clinical evidence of dehydration. Examination of the ears, nose, throat, lungs and heart revealed nothing abnormal. The abdomen was soft and not distended. There were no palpable masses. Peristalsis was not audible. The extremities were normally formed and exhibited good tone.

Shortly after admission the patient passed his first tenacious meconium plug. His diet consisted of 5% *Beta*-lactose in one-half ounce portions every four hours, given by a nipple at first, later by gavage. All the fluid ingested was regurgitated as a thick brownish liquid. Elixir of phenobarbital gr. 1/12 and atropine sulfate gr. 1/1,000, given ten minutes before each feeding, failed to prevent the vomiting. Hypodermoclyses of 50 cc. of physiologic saline solution were administered every six hours and Klotogen in 0.5 cc. doses was given every eight hours.

A surgical consultation was held ten hours after the infant arrived at the hospital. At that time the child presented a strange and frightful picture. Streams of fresh blood came from the nostrils and mouth, while the lips and chin were coated with dried blood. Immediately before the consultation the child had vomited two ounces of bright blood. The skin was mildly dehydrated. Pinching the skin elicited a lusty cry. The abdomen was soft but slightly distended. The liver edge was easily palpable, extending from the xiphoid process to the right anterior superior iliac spine. An indefinite tubular structure was palpable extending from the midpoint of the left costal margin downwards towards the umbilicus. Peristaltic waves were not visible.

The diagnoses listed in order of preference were: 1. Duodenal obstruction, either extrinsic or intrinsic; 2. volvulus; and 3. an hemorrhagic diathesis, together with some form of high intestinal obstruction.

On March 8, 1941, 24 hours after admission, a roentgenographic examination was performed. Fluoroscopically, the chest, heart and diaphragm appeared normal. A small amount of barium passed unimpeded down the esophagus, outlined the stomach normally, and then passed through the pylorus. The barium collected in the first portion of the duodenum and had remained there at the time of the second examination 24 hours later. *Roentgenologic Diagnosis:* Duodenal obstruction.

Operation was performed March 9, 1941, at 4 P.M. Preoperative medication consisted of sodium phenobarbital gr. ¼ hypodermically at 3:15 P.M. Analgesia was ob-

tained by means of a whiskey sugar-teat, and anesthesia by 1% procaine infiltration. A right rectus muscle-splitting incision was made.

Examination revealed a dilatation of the pylorus to 1.5 cm., and a dilatation of the first portion of the duodenum to 3.0 cm. At the line of juncture with the second portion of the duodenum this dilatation of the first portion ended abruptly in a rounded, blind sac. A hiatus, of about 2 Mm. in width, separated the first and second portions of the duodenum. The second portion was slightly distended and was tinged green-black by the bile which it contained. Examination of the stomach, jejunum, ileum, and colon revealed no gross abnormalities.

A duodenojejunostomy was effected between the dilated first portion of the duode-

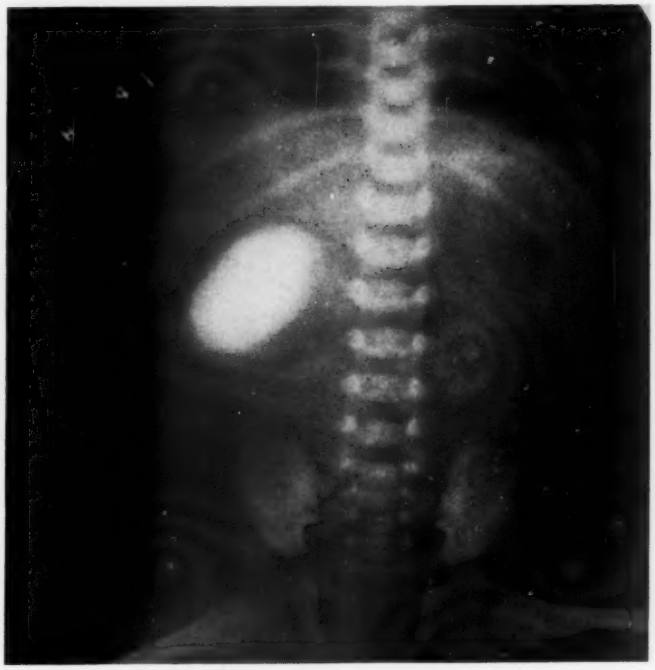


FIG. 1.—Roentgenogram showing the dilated first portion of the duodenum filled with barium, 24 hours after barium was ingested.

num and a loop of jejunum about 10 cm. distal to the ligament of Treitz. The anastomosis was held by means of an outer row of interrupted and an inner row of continuous sutures, posteriorly, and a Connell suture and outer row of interrupted sutures, anteriorly. Arterial silk No. 00000 was used throughout.

It was necessary to induce light ether anesthesia to close the abdomen. The peritoneum and rectus sheath were closed as one layer with closely placed interrupted sutures of No. 00 silk. Interrupted vertical mattress sutures of No. 00 silk approximated the skin edges.

A transfusion of 70 cc. of citrated blood was administered immediately after operation. The postoperative reaction was negligible. Aspiration of the stomach contents every four hours, during the first 48 hours, yielded 2 to 8 cc. of bile-colored fluid. Sterile water was given in amounts not exceeding 15 drops every two hours, and 50 cc. saline infusions were continued, hypodermically, every six hours. Sulfanilamide, 15 cc. of 0.8% solution, was added to each infusion.

Twenty-four hours after operation the infant became cyanotic and developed rapid, irregular breathing. Simultaneously, tetanic contractions of the hands occurred. These phenomena were relieved by inhalations of oxygen and intramuscular injections of

calcium gluconate. Physical examination of the lungs revealed no abnormal signs.

On the second day after operation there were several short-lived attacks of respiratory difficulty which were relieved by oxygen inhalations.

On the third day ten drops of 5% glucose solution, alternating with ten drops of mother's milk, were taken every hour. Regurgitation of indeterminate amounts of bile-colored fluid occurred. A dark green stool indicated that bile was passing through the anastomosis. A urine specimen gave a strongly positive reaction for acetone. To combat this acidosis 2.5% glucose solution was added to the saline infusions. A second transfusion of 70 cc. of citrated blood was administered. The temperature dropped to 99.8° F. at the end of the third day.

On the fourth day a disruption of the central two-thirds of the wound was discovered, with the anterior surface of the right lobe of the liver filling the gap. The skin edges were approximated with strips of adhesive plaster and a vaselined gauze dressing was applied. Resuture was postponed in the hope that improvement in the general condition might occur as the function of the duodenojejunostomy became established. The child was taking 160 cc. of milk and glucose solution by mouth in 24 hours, and regurgitating about one-half this intake. The temperature remained normal on the fourth day and until noon of the fifth day postoperatively.

The volume of fluid regurgitated diminished on the sixth day and the infant appeared stronger. Therefore, the wound was resutured under light ether anesthesia. Interrupted through-and-through sutures of No. 38 steel wire, extending from skin to peritoneum, were placed in the abdominal wall. Skin edges were carefully approximated with interrupted sutures of No. 40 steel wire. The operation required 37 minutes.

The infant appeared to be severely shocked immediately following the operation, and after 45 minutes respiration became imperceptible but was restored with the aid of a resuscitator. The improvement thus obtained was short-lived. Repeated resuscitation was necessary until death occurred two hours and 45 minutes after operation, March 15, 1941.

Autopsy.—This was performed 20 hours postmortem. The cause of death was lobular pneumonia, involving the lower lobes of both lungs. Microscopic examination showed this process to be in the stage of grey hepatization.

Pathologic Examination.—Gross: The stomach was long and of the fish-hook type. The duodenal bulb was dilated, and a complete atresia was found 3 cm. distal to the pylorus. There was no abnormality of the common bile duct which emptied into the second portion of the duodenum just distal to the line of atresia *via* a dilated ampulla of Vater.

The duodenojejunal anastomosis was intact and there was no evidence of serosal irritation along the suture line. *Microscopically,* numerous fibroblasts were seen in the inflammatory exudate which cemented the serosal surfaces. This degree of healing appeared to be normal for the elapsed time.

SUMMARY

This is a report of a case of congenital atresia of the duodenum in an infant 30 hours old. Roentgenologic examination proved the atresia to be complete. A duodenojejunostomy was performed 50 hours after birth. The child had attacks of respiratory difficulty which recurred with increasing frequency after the second postoperative day. On the fourth day postoperatively the wound was found disrupted. Liquids taken by mouth were retained in one-half to one-third the ingested amounts, and there was evidence that the anastomosis functioned. The wound of the abdominal wall was resutured on the sixth day following the first operation. Three hours later the infant died. Autopsy showed the anastomosis to be intact. Bilateral pneumonia was the cause of death.

MULTIPLE ATRESIA OF THE SMALL INTESTINE

CASE REPORT

D. M. GLOVER, M.D., SIMMONS SMITH, M.D.,

AND

OLIVER EITZEN, M.D.

CLEVELAND, OHIO

FROM THE DEPARTMENTS OF SURGERY AND PATHOLOGY OF CLEVELAND CITY HOSPITAL, AND THE MEDICAL SCHOOL OF WESTERN RESERVE UNIVERSITY, CLEVELAND, OHIO

OF THE MANY CAUSES of intestinal obstruction in the newborn, multiple atresia is one of the rarest. The case presented herewith, which showed eight separate and complete points of occlusion of the small intestine was observed at Cleveland City Hospital. From a cursory review of the literature, it seems probable that fewer than 100 such cases have been reported.

Webb and Wangensteen[1] estimated that some form of congenital obstruction of the bowel is found approximately once in 20,000 births. Ladd[2] reported 60 cases of obstruction due to intestinal anomalies; of these 40 were due to intrinsic defects. Glover and Hamann[3] reviewed the records of three large general hospitals in Cleveland, covering a 17-year period, and were able to collect 18 cases of acute intestinal obstruction due to anomalies; of these, five showed points of atresia. Davis and Poynter,[4] who studied a large series of cases in 1922, believe that in approximately 15 per cent of the atretic cases the points of atresia are multiple.

Case Report.—Baby G., white, female, was born August 18, 1940 at 1:00 P.M., about two months prematurely. Previous to admission to the hospital the baby was kept at home in a basket, without any form of external heat. On the day after birth, following the mother's attempt to give her some boiled water, she vomited a small amount of bile-stained fluid. Until admission to the hospital, August 20, 1940, almost two full days after birth, she had retained no significant amount of water or milk. There had, furthermore, been no stool or passage of meconium.

At the time of admission, physical examination showed a premature, moderately icteric infant, whose temperature was 35°C. Following each feeding she vomited bile-stained fluid, and peristaltic waves passing from left to right could be easily seen in the epigastric region. Several small enemata were given, following which only colorless mucus was passed. Roentgenologic studies, made on the morning following admission, showed the stomach and duodenum to be greatly distended with gas, but no gas was visualized in the remainder of the small intestine or colon (Fig. 1). After the administration of a small amount of a barium suspension by mouth, the dilated stomach was readily filled by the contrast medium, as were the first and second parts of the duodenum. The third part of the duodenum (as shown in the oblique views) seemed to lie anterior to and below the stomach, and then appeared to pass about the greater curvature of the stomach to the left and come to a blunt point of occlusion behind the stomach. Beyond this point neither gas nor barium would pass. The interrugal folds of the duodenum were readily visualized (Fig. 2).

Combining the history of vomiting bile-stained fluid from the time of the first feeding, the physical evidence of dilatation and visible peristalsis in the stomach, and the roentgenographic evidence of distal duodenal obstruction, with anterior position of the duodenum, the operator (D. M. G.) thought that the evidence favored a diagnosis of unrotated colon with volvulus of the small intestine about the superior mesenteric artery. With this preoperative diagnosis, an exploratory celiotomy was performed, under local anesthesia, through a small upper right transverse incision.

Operative Pathology: Upon opening the peritoneum, an enormously distended loop of intestine presented. The stomach and duodenum were markedly distended, with flecks of barium visible through the thinned-out visceral wall. The duodenum, which was about 2 cm. in diameter, occupied an anterior position below the stomach, in front of

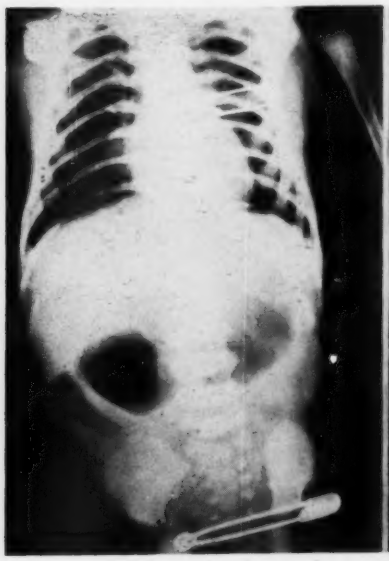


FIG. 1.—Roentgenogram showing the distended, gas-filled stomach and duodenum.

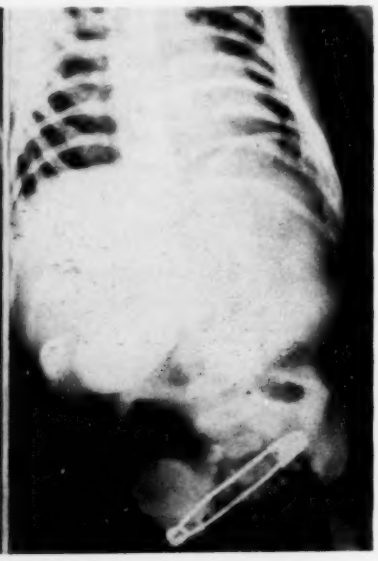


FIG. 2.—Roentgenogram showing stomach and duodenum partly filled with barium and gas. Neither barium nor gas can be seen beyond the distal duodenum, which clearly shows interrugal folds.

the superior mesenteric artery, curving to the left behind the stomach and ending blindly at a point, roughly, in the position of the duodenojejunal junction. The cecum and appendix were just to the left of the midline below and behind the duodenum. The distal ileum entered the cecum from right to left, and the entire colon occupied the left side of the abdomen, in unrotated fetal position. The small intestine was collapsed, very small in caliber, and occupied the right lower abdomen. It was impossible, without completely eviscerating the infant, to determine the exact position of the proximal jejunum. A loop of small intestine, which seemed large enough to make an anastomosis possible, but which was actually only about 4 mm. in diameter, was anastomosed to the blind end of duodenum, by the end-to-side method, using fine silk sutures throughout, without the use of clamps. Gas and fluid passed immediately into the small intestine from the duodenum. The abdominal wall was closed in layers with fine catgut.

Following operation the infant began taking a small amount of formula every hour, and regurgitated only a little. On the second postoperative day a small, yellow stool was passed. On the third postoperative day (August 23, 1940), however, the infant became weak, cyanotic, and died.

Autopsy.—Gross Pathology: External examination revealed moderate icterus. There was a healing scar in the right upper quadrant, 4 cm. in length. No anomalies of the external surface, head or extremities were noted.

Section revealed the thoracic and abdominal organs, other than the intestinal tract, to be in the usual position. The stomach was slightly distended. The intestinal tract showed complete lack of rotation. The entire duodenum was markedly dilated. The

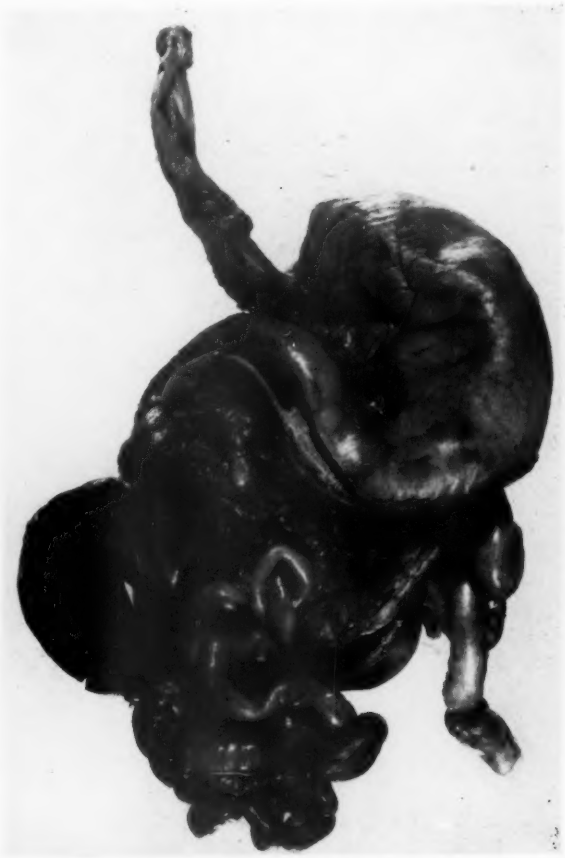


FIG. 3.—The postmortem appearance of the abdominal viscera. The distended duodenum may be seen on the left anastomosed to the ileum. The cecum and appendix are in the midline below the stomach.

first and second portions were in the usual position but the third, instead of passing over the vertebral column in the usual manner, passed downward on the right side of the abdomen and, at the height of the umbilicus, turned cephalad into the epigastrium and crossed the spinal column in its terminal portion to end blindly. A loop of ileum 8 cm. from the ileocecal junction had been attached in side-to-end anastomosis to this blind end. The jejunum began in the root of the mesentery as a filamentous strand 1 mm. in diameter, gradually widened to an average diameter of 5 to 8 mm., then terminated abruptly in a blind end. In the remaining distal portion of the jejunum and ileum seven additional areas of atresia were found (Figs. 3 and 4). The segments of intestine between these atretic areas varied from approximately 2 to 5 cm. in length, were

cylindrical, and had bluntly rounded ends. The portions of the mesentery between the segments ended in slightly thickened, rounded free edges. The entire root of the mesentery was on the left side of the spine. The final segment of the small intestine measured about 18 cm. and emptied into the cecum which was found in the left upper quadrant. The appendix was normal. The colon extended by a free mesenteric attachment in the left colic gutter to the pelvis where it terminated in the sigmoid and rectum in the usual manner. The serosal surfaces in the region of the anastomosis were covered by a light reddish-brown fibrinous exudate. The stoma of the duodeno-ileostomy was very small but patent, and no obstruction in the intestinal tract distal to the anastomosis was found.

FIG. 4.—The postmortem specimen showing seven areas of atresia in the small intestine.

Microscopic: The segments of the intestine ended in normal mucosa and submucosa. However, as shown by serial sections, both the longitudinal and circular muscle layers of the intestine, diminished in size, continued across the gaps in the mesentery and linked succeeding segments. This muscle showed no microscopic abnormalities other than small size of the layers. Cross-sections of the portions of mesentery between the blind ends of intestine showed muscle fibers, longitudinally and circularly disposed, surrounding a mass of connective tissue either completely or partially, the mesenteric side being composed of loose vascular connective tissue. A section of the duodenojejunostomy showed edema, hyperemia, mild acute inflammation, necrosis, clusters of cocci, and amorphous masses. These changes were more marked in the duodenal than in the ileal wall.

Sections of lungs, liver, spleen and kidneys showed moderate passive hyperemia. A few small focal hemorrhages were seen in the lower lobe of the left lung and in the pyramids of the kidneys.

REFERENCES

[1] Webb, C. H., and Wangensteen, O. D.: Congenital Atresia of Intestines. Am. Jour. Dis. Child., **41**, 262-284, 1931.

[2] Ladd, W. E.: Congenital Obstruction of the Small Intestine, J.A.M.A., **101**, 1453-1458; Surgical Diseases of the Alimentary Tract of Infants. New Engiand J., **215**, 705, 1936.

[3] Glover, D. M., and Hamann, C. A.: Intestinal Obstruction in the Newborn. Ohio State Med. J., **36**, 833-840, 1940.

[4] Davis, D. L., and Poynter, C. W. M.: Congenital Occlusion of the Intestines: Report of a Case of Multiple Atresia of the Jejunum. Surg., Gynec. and Obst., **34**, 35, 1922.

A LARGE SUBACUTE GASTRIC ULCER

CASE REPORT

MAJOR JAMES B. MASON, M.C.,

CHIEF, SURGICAL SERVICE

1ST LT. ERWIN D. ZEMAN, M.C.

SECTION ON PATHOLOGY

STATION HOSPITAL, CAMP LEE, VA.

MOST GASTRIC ULCERS seen in reported autopsy and surgical material are chronic or of the acute perforating variety and conform to the usually described round or ovoid ulcer of Cruveilhier.[1, 2, 3] Descriptions and illustrations of gastroscopic studies[4] made during the progress and development of gastric ulcers do not alter this general impression.

The following is reported chiefly to illustrate the unusual form of a large subacute ulcer, surgically removed because of hemorrhage and obstruction, thought to be due to gastric malignancy. The pattern of the ulcer bed and its apparent coincidence with the distribution of the subserosal blood vessels of the lesser curvature of the stomach is likewise considered noteworthy.

Case Report.—E. D., Negro, male, age 51, was admitted to the Station Hospital, Camp Lee, Va., November 10, 1941, with the chief complaint of vomiting "coffee ground" material. Prior to admission, October 31, 1941, he had first complained of loss of appetite and "growling" in his stomach. He had taken three compound cathartic pills and had had three copious bowel movements containing mucus but no gross blood. He was unrelieved of the cramping pain but the catharsis and the anorexia persisted. On November 2, he vomited three or four times. During the next week he was unable to retain solid food but could take liquids. Severe cramping pains coming in waves, preceded the vomiting. He had not had a bowel movement since November 1, although he had passed flatus. His past history revealed normal bowel function until October 31, 1941. He had had malaria in childhood. Operations had included the excision of a lipoma in 1929 and 1931, and the excision of a fistula in ano in 1931. He was unmarried, denied venereal infection, used alcohol moderately and smoked 10 to 12 cigarettes daily.

Physical Examination.—The patient was a well-nourished male, height 71 inches, weight 210 pounds. He was acutely ill. Positive findings were moderate pallor of mucous membranes of the mouth and lips, and slight midepigastric tenderness, but no rigidity. No adbominal masses, tympanites nor increased intestinal activity were noted. There were a few small, shotty inguinal lymph nodes. His heart and lungs were negative. Blood pressure 124/70 mm. Reflexes normal.

Laboratory Data.—R. B. C. 5,135,000-4,600,000. W. B. C. 21,000-12,000. The high initial counts were undoubtedly due to dehydration. Hb, 95-80 per cent (Tallqvist). The blood Kahn was positive on November 17, and again on November 27, 1941. Gastric Analysis.—November 12, 1941: Total acidity of 75 units, and a free hydrochloric acid of 45 units. Pus cells and R. B. C. were abundant. Roentgenologic examination, November 13, 1941, revealed an "ulcer niche of the stomach on the superior margin of the pyloric area with evidence of infiltration which is strongly suspicious of malignancy." *Preoperative Clinical Diagnosis.*—Carcinoma of the stomach with obstruction.

Preoperative Course and Management.—General supportive treatment was instituted, including gastric drainage by the method of Wangensteen, and water electrolyte balance was controlled by the intravenous route. During this period there was considerable gastric distress and the drainage continued to contain blood. The patient steadily lost in strength and weight, as it was impossible to pass the duodenal tube beyond the pylorus and into the duodenum for feeding purposes, due to the apparent obstruction. He had two blood transfusions of 500 cc. of whole citrated blood. Early nutritional edema developed on or about November 17th, so that it was decided that surgical intervention was necessary despite the inability to improve his nutrition.

Operation (J. B. M.).—November 19, 1941: Under general anesthesia (gas induction open drop-ether), a large ulcer-bearing area of the pyloric region was found, with greatest tumefaction and hyperemia at the lesser curvature and extending almost to the greater curvature on both gastric walls. There were no adhesions or enlargement of the regional or mesenteric lymph nodes, neither did exploration reveal nodules of the liver or retroperitoneum. A subtotal resection was accomplished by removing between one-third and one-half of the stomach. Intestinal continuity was reestablished by the anterior Pólya method. A Witzel-type jejunostomy for feeding purposes was established in the proximal loop of the jejunum about four inches below the anastomosis. The catheter was threaded through the mesentery and brought through a separate stab incision to the left of the exploration incision, which was an upper left rectus type. One of us (J. B. M.) has for a number of years adopted this procedure as a part of the operative technic for postoperative feeding,[5] with substantial benefit to all patients so treated.

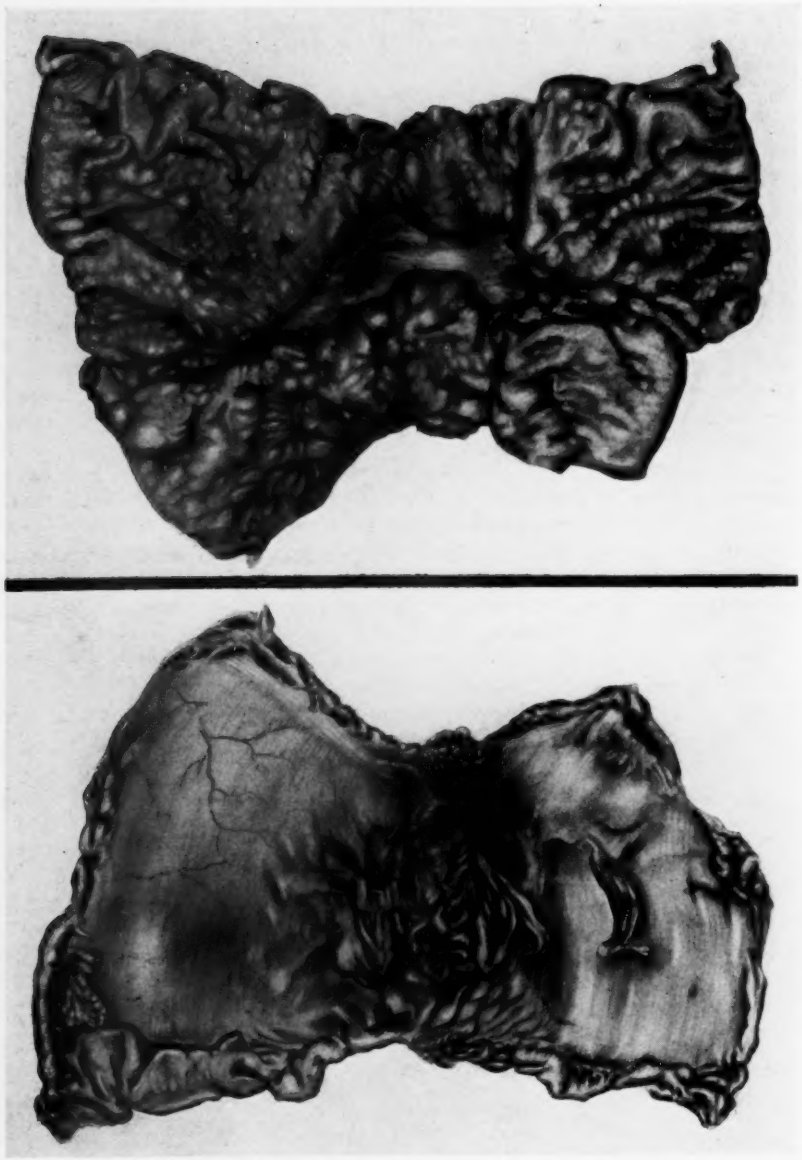
Postoperative Course.—The postoperative course was exceedingly smooth. Of especial interest was the feeding problem which was made simple by the jejunostomy. For a number of years the formula of Scott and Ivy [6] had been used with benefit, but in this instance the simplified formula recommended by Clute and Bell [7] was found to be entirely adequate, and in a military hospital has much to recommend it because of the simplicity of preparation. The hydration and nutrition of the patient immediately improved, and by the fourth postoperative day he stated that he felt strong enough to get out of bed. The jejunal feedings were continued for a week (about 2500 calories per day, with adequate protein, fat, carbohydrate, electrolyte and vitamins being given each 24 hours by a feeding schedule which continued through the day, save from 10 P.M. to 4 A.M.). The apparatus used was one previously described,[5] and consists of a narrow blood burette with a controlled Murphy drip in the system to the catheter. Too rapid flow caused increased peristalsis with pain and diarrhea (about one hour was allowed to introduce the 250 cc. feeding to the jejunum). The formula of Clute and Bell has the further advantage that it is low in fat. The tendency to diarrhea noted in other feeding formulas was absent. Supplemental feedings by mouth were instituted after the first week, and on the twelfth postoperative day the jejunal tube was removed.

A gastric analysis, December 10, 1941, revealed a total acidity of 15 units, but no free hydrochloric acid. Antiluetic therapy was also instituted at this time.

A generous diet was ordered and he had regained his strength by December 15, 1941, when he was discharged.

Pathologic Examination.—Gross.—The truncated horn-shaped specimen consisted of the amputated distal portion of the stomach, and measured eight centimeters along the lesser curvature and 12 cm. along the greater curvature. The diameter of the fundus portion was seven centimeters, while that of the pyloric end was four centimeters. The amputation margins had been crushed. The peritoneal surface was moderately hyperemic and there were numerous petechial hemorrhages in the edematous fatty mesentery of the lesser curvature. Many catgut ligatures were present on the curvatures. When spread out, the excised portion measured roughly 16 x 10 cm. There was an extensive saddle- or girdle-like ulceration centered at the lesser curvature, three centimeters from the

FIG. 1.



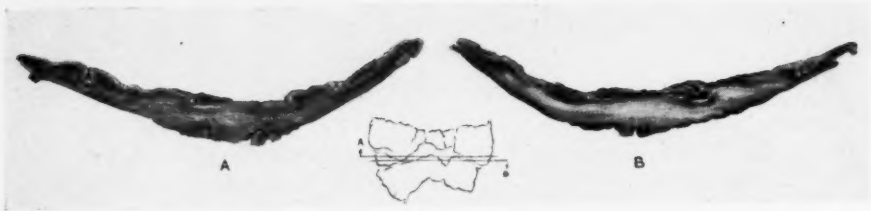


FIG. 2.

FIG. 1.—(A) Drawing of pyloric portion of resected stomach including the ulcer. The shape and extent of the ulcer pattern in comparison with the vascular distribution on the serosal surface (B) is noteworthy. The prominence of the eroded blood vessels is exaggerated in the drawing. FIGURE 2 shows the gross characteristics of longitudinal sections of the two edges of the ulcer. (Drawings by Pvt. Raphael Epstein, Med. Dept. U. S. Army.)

pyloric amputation level, extending circumferentially four centimeters along the anterior and 4.5 cm. along the posterior wall. It tapered from 1.5 cm. in its widest portion at the lesser curvature to narrow grooves extending to within two centimeters of the greater curvature on both walls. The margins of the ulceration were rolled and smooth. There was considerable edema and hyperemia including petechial hemorrhages in a region two to three centimeters on either side of the ulceration at the lesser curvature. There was little induration. The flat ulcer floor was eight millimeters below the mucosal margins. The surface was granular with fibrin, clotted blood and glairy mucous, which was readily wiped off. In the wide portion of the ulcer two blood vessel stumps protruded about 0.5 mm. above the ulcer bed. These had an outside diameter of one millimeter. The lumens contained clotted blood (Figs. 1 and 2).

Microscopic.—The mucosa at some distance from the margin of the ulcer was composed of slightly hypertrophic glands between which were scattered moderate numbers of plasma cells. A few solitary lymphoid follicles lying in the deeper parts of the mucosa were considerably enlarged and had conspicuous reaction centers. At the margin of the ulcer, the mucosa, rather abruptly, became thinner, the glandular structures were lost until, at the very edge of the ulcer, a single layer of epithelial cells formed the sole remnant of the mucosa.

The surface of the ulcerated area consisted of clotted plasma in which lay degenerating leukocytes and cell detritis. The entire gastric wall beneath the ulcer, as well as beneath the still preserved mucosa, was edematous and everywhere infiltrated by leukocytes, lymphocytes, plasma cells and histiocytes. The muscle had been replaced, in some areas, by granulation tissue, composed of newly-formed capillaries and fibroblasts in various stages of maturity. Here and there, but particularly in the deeper portions of the wall, was an abundant fatty areolar tissue. In the deeper parts there are also numerous nerve bundles and small nerve ganglia, most of which were imbedded in the inflammatory reaction which extends to the serosa.

In a section from a portion near the ulcer, the surface was covered with gastric mucosa which in one region was thickened to nearly twice normal. This region was sharply delineated and consisted of normally arranged, slightly hypertrophic glandular tissue, with much lymphoid hyperplasia. The submucosa contained fatty-areolar tissue and presented inflammatory reaction similar to that described above. There were a few small hemorrhages. In none of the sections was there any evidence of malignancy. There was no indication from a study of the blood vessels, of vascular alteration of syphilitic or sclerotic nature. Preparations from the ulcer margins of the unfixed fresh tissues examined by darkfield illumination failed to reveal spirochaetal forms. *Pathologic Diagnosis.*—Subacute ulcer of the stomach with marked inflammatory reaction (Figs. 3 and 4).

COMMENT.—This large subacute gastric ulcer of unusual shape presents several interesting features from a pathologic as well as clinical viewpoint.

The lack of specific alterations of the vascular tissues, the absence of patchy scarring, the failure to find spirochetes in the ulcer margins, and the high gastric acidity, unusual in reported gastric syphilis,[8] would seem to eliminate syphilis as a causative factor in spite of the positive Kahn reaction.

Numerous writers have been concerned with the relationship of the vascular supply of the lesser curvature with the coincidence of ulcer.[9] Whether this relationship is significant in the case here reported cannot certainly be ascertained, but the conformity of the lesion to the subserosal vascular pattern of the lesser curvature seems especially noteworthy (Figs. 1 and 2).

The treatment was obviously surgical, to rule out malignancy. The

operation was performed while the nutrition of the patient was impaired by inability to pass a feeding tube beyond the site of obstruction. Operation revealed that this failure to pass the duodenal tube was due to the severity of pyloric spasm.

FIG. 3.

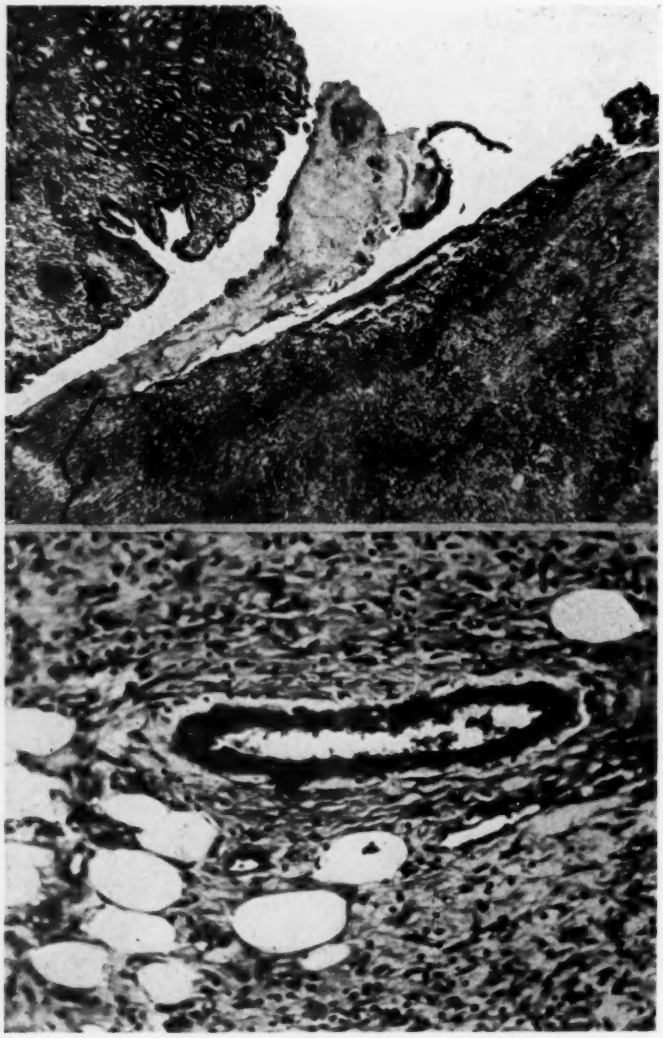


FIG. 4.

FIG. 3.—Photomicrograph including a margin of the ulcer. Note the narrow layer of epithelial cells extending to the ulcer floor.

FIG. 4.—Photomicrograph of a section in the ulcer floor, including small blood vessel, without unusual change. Note cellular distribution.

The treatment by medical means might have been attempted but for the two just mentioned reasons. One may speculate as to the subsequent course of this lesion, and its ultimate status—whether complete healing without scarring might occur, or, the far greater likelihood, that had time inter-

vened, that a chronic type of circumscribed indurated ulcer would have eventuated. Thus does one wonder that here, due to impairment of a vascular area, does not necrosis of tissue occur, with loss of tissue substance in the mucosal area so supplied, with reaction and healing of a portion save that where too great a mucosal loss has occurred, and here, due to high gastric acids plus aging of repair, a chronic ulcer of the lesser curvature might not have occurred.

The end-result was satisfactory, in that the man was doing light work two months after discharge from the hospital.

REFERENCES

[1] Robertson, H. E.: In Eusterman and Balfour. The Stomach and Duodenum. W. B. Saunders Co., 1935, p. 97.

[2] McCarty, W. C.: *Ibid*, p. 78.

[3] Cecil, R. L.: A Textbook of Medicine. Ed. 5, W. B. Saunders Co., 1941.

[4] Schindler, R.: Gastroscopy: The Endoscopic Study of Gastric Pathology. University Chicago Press, 1937.

[5] Mason, James B.: Jejunal Feedings following Operations on the Stomach and Duodenum. Penna. Med. Jour., **41**, 1083-1086, September, 1938.

[6] Scott, H. G., and Ivy, A. C.: Jejunal Alimentation: An Experimental Study in Dogs. ANNALS OF SURGERY, **93**, 1197-1201, June, 1931.

[7] Clute, H. M., and Bell, L. M.: Jejunostomy for Postoperative Feeding. ANNALS OF SURGERY, **114**, 462-471, September, 1941.

[8] Williams, C., and Kimmelstiel, P.: Syphilis of the Stomach, J.A.M.A., **115**, 578-582, August 24, 1940.

[9] Reeves, T. B.: Studies of Arteries Supplying the Stomach and Duodenum and their Relation to Ulcer. Surg., Gynec. and Obst., **30**, 374-385, 1920.

operation was performed while the nutrition of the patient was impaired by inability to pass a feeding tube beyond the site of obstruction. Operation revealed that this failure to pass the duodenal tube was due to the severity of pyloric spasm.

FIG. 3.

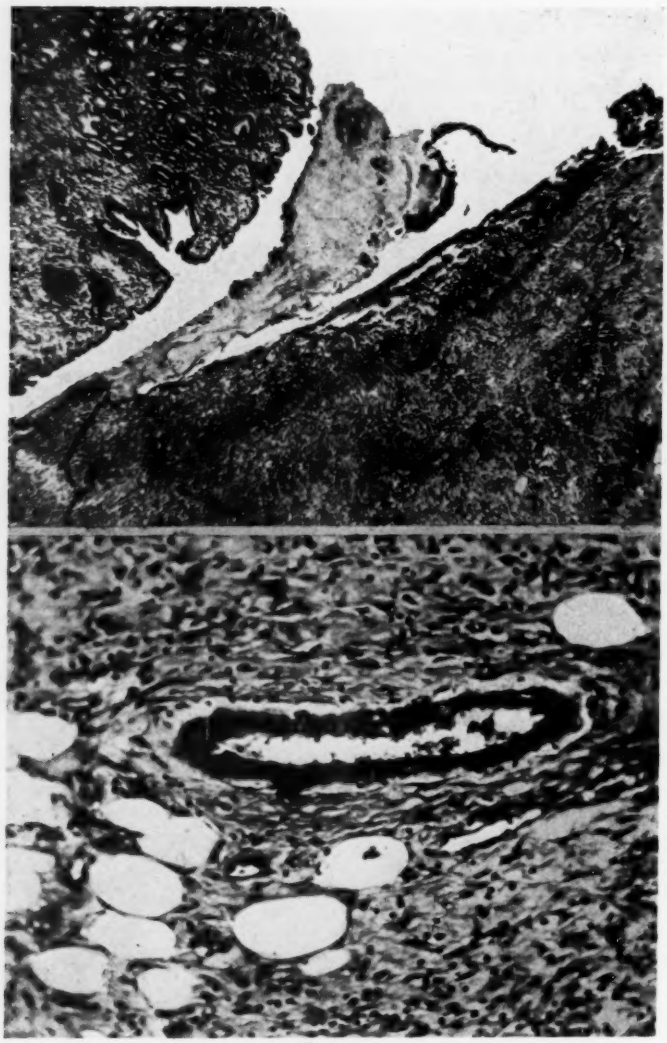


FIG. 4.

FIG. 3.—Photomicrograph including a margin of the ulcer. Note the narrow layer of epithelial cells extending to the ulcer floor.

FIG. 4.—Photomicrograph of a section in the ulcer floor, including small blood vessel, without unusual change. Note cellular distribution.

The treatment by medical means might have been attempted but for the two just mentioned reasons. One may speculate as to the subsequent course of this lesion, and its ultimate status—whether complete healing without scarring might occur, or, the far greater likelihood, that had time inter-

vened, that a chronic type of circumscribed indurated ulcer would have eventuated. Thus does one wonder that here, due to impairment of a vascular area, does not necrosis of tissue occur, with loss of tissue substance in the mucosal area so supplied, with reaction and healing of a portion save that where too great a mucosal loss has occurred, and here, due to high gastric acids plus aging of repair, a chronic ulcer of the lesser curvature might not have occurred.

The end-result was satisfactory, in that the man was doing light work two months after discharge from the hospital.

REFERENCES

[1] Robertson, H. E.: In Eusterman and Balfour. The Stomach and Duodenum. W. B. Saunders Co., 1935, p. 97.
[2] McCarty, W. C.: *Ibid*, p. 78.
[3] Cecil, R. L.: A Textbook of Medicine. Ed. 5, W. B. Saunders Co., 1941.
[4] Schindler, R.: Gastroscopy: The Endoscopic Study of Gastric Pathology. University Chicago Press, 1937.
[5] Mason, James B.: Jejunal Feedings following Operations on the Stomach and Duodenum. Penna. Med. Jour., **41**, 1083-1086, September, 1938.
[6] Scott, H. G., and Ivy, A. C.: Jejunal Alimentation: An Experimental Study in Dogs. ANNALS OF SURGERY, **93**, 1197-1201, June, 1931.
[7] Clute, H. M., and Bell, L. M.: Jejunostomy for Postoperative Feeding. ANNALS OF SURGERY, **114**, 462-471, September, 1941.
[8] Williams, C., and Kimmelstiel, P.: Syphilis of the Stomach, J.A.M.A., **115**, 578-582, August 24, 1940.
[9] Reeves, T. B.: Studies of Arteries Supplying the Stomach and Duodenum and their Relation to Ulcer. Surg., Gynec. and Obst., **30**, 374-385, 1920.

MULTIPLE, PRIMARY NONSPECIFIC JEJUNAL ULCERS, WITH CHRONIC DUODENAL DILATATION

EDWARD DOWDLE, M.D.

DETROIT, MICH.

FROM THE PROVIDENCE AND ELOISE HOSPITALS, DETROIT, MICH.

PRIMARY nonspecific jejunal ulcer and chronic duodenal ileus are both rather rare, but important lesions. In the present instance the chronic duodenal ileus was a complication of jejunal ulcer.

Case Report.—N., white, male, age 59, was admitted to Providence Hospital, December 11, 1940, with the history that he had, during the last ten years, passed "black stools" on numerous occasions and had occasional spells of "indigestion and dyspepsia," for which he never consulted a physician. The past history is otherwise irrelevant.

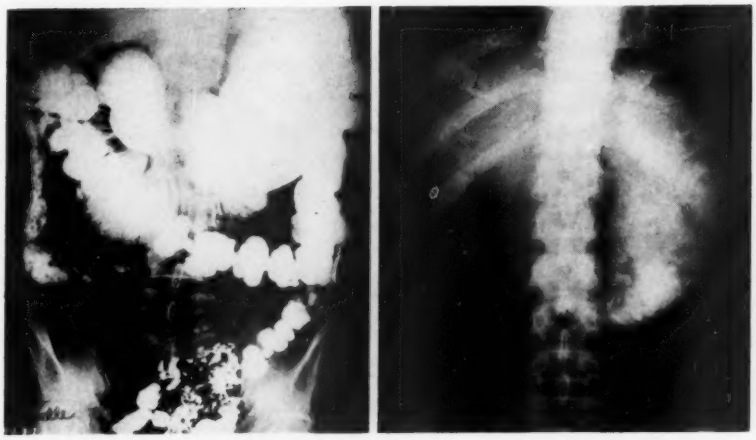
For six months he had vomited one to two hours following meals. At first the vomiting occurred at infrequent intervals and was not in large amounts but grew progressively worse, occurred more frequently, in larger amounts, and was often of greenish color. For several months he has vomited after each meal and even between meals. He has had no epigastric pain nor distress other than a feeling of fullness, which has always been relieved by vomiting.

During the last six months he had lost over 40 pounds in weight, became considerably weakened, and was always moderately constipated. On physical examination he was well-developed but undernourished and dehydrated. His tongue was dry and coated. There was a soft mitral systolic murmur over the apex of the heart and some moist râles at the bases of both lungs. The abdomen was soft, moderately distended and tympanitic. There were no masses or areas of tenderness. Blood pressure 120/60; temperature, pulse and respirations normal.

Laboratory Data: R.B.C. 5,160,000, hemoglobin 112 per cent; W.B.C. 8,000, with neutrophils 78 per cent, lymphocytes 18 per cent and monocytes 4 per cent. Urine: Trace of albumin, and 20 to 25 leukocytes per high power field. Roentgenologic examination demonstrated an obstruction of the small bowel in the region of the ligament of Treitz (Fig. 1A). This produced almost complete obstruction, with a marked dilatation of the entire duodenum and stomach. One week later there was still a minimum amount of barium in the stomach, indicating marked gastric delay (Fig. 1B). A barium enema showed no abnormalities. *Clinical Diagnosis:* Chronic duodenal ileus due to obstruction near the duodenojejunal angle. The obstruction was thought to be an annular carcinoma or ulcer.

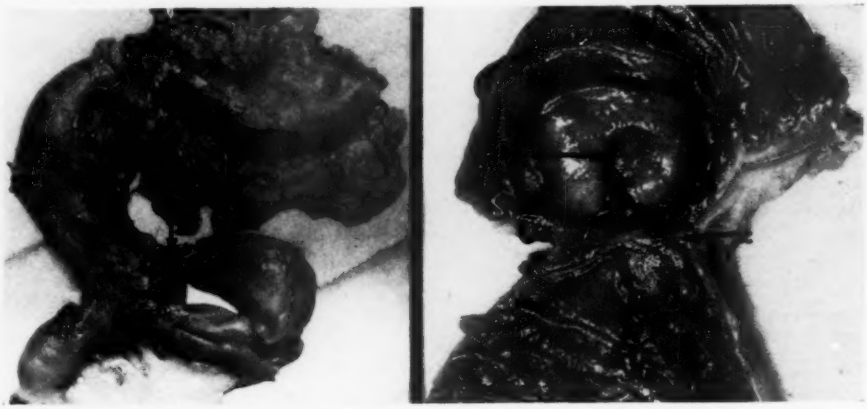
He was prepared for operation from December 11 to December 20, 1940. Wangensteen suction drainage was instituted to decompress the stomach and duodenum. Fluid was administered parenterally as 1000 cc. of 10 per cent glucose in saline every six hours, and a liquid diet was given. On the day before operation, he received CO_2 inhalations, digalin intramuscularly and three 5 mg. doses of cortate. Whole blood chloride on this day was 483 mg. per cent, and nonprotein nitrogen 26.9 mg. per cent.

Operation.—December 20, 1940: Under continuous spinal anesthesia, the abdomen was opened through a midline incision. The stomach and first part of the duodenum were quite dilated. The jejunum, about one inch below the ligament of Treitz, was markedly adherent to the root of the mesentery in the region of the superior mesenteric vessels and, at this point, seemed to be completely constricted. The jejunum above this point was dilated and the wall thick and edematous. The lymph nodes in the adjacent mesentery were markedly enlarged. One, removed for biopsy, proved to be inflammatory.



(A) (B)

FIG. 1.—(A) Note dilatation of entire duodenum secondary to obstruction near the ligament of Treitz. (B) Plain film of abdomen one week after barium meal, showing barium still present, indicating the completeness of the obstruction.



(A) (B)

FIG. 2.—(A) The stomach and duodenum are dilated but not so markedly as at the time of operation. An arrow indicates the point of stricture. (B) Arrow 1 indicates the annular, and arrow 2 the circular ulcer. It was the former which produced the constriction.

To accomplish a short-circuiting operation, it was necessary to divide the ligament of Treitz. This allowed mobilization of almost three inches of jejunum and duodenum. A lateral duodenojejunostomy around the obstruction was easily accomplished. Silk was used for the outer layer. Three finger tips could be inserted into the stoma. At the end of the operation the patient began to strain, vomited considerable bile, and became very cyanotic.

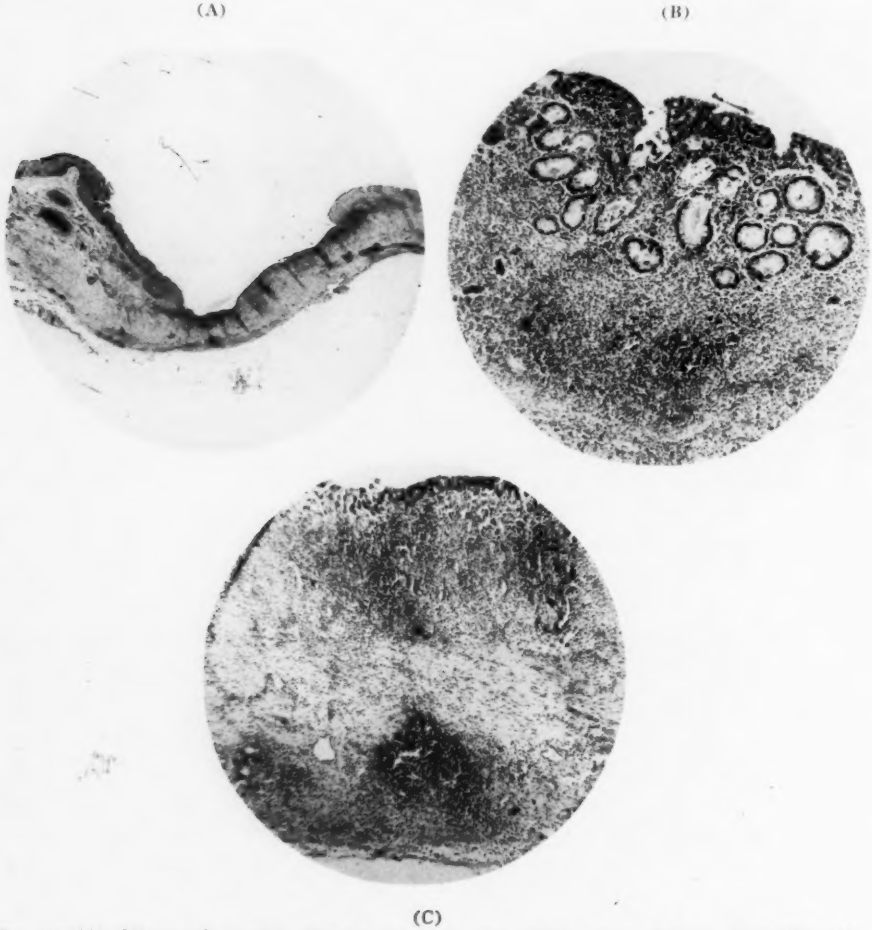


FIG. 3.—(A) Microscopic sections through the ulcer area—whole section through ulcer (Fig. 2). (B) The border of the ulcer. (C) The ulcer base.

Postoperative Course.—Within 24 hours following the operation the temperature was 106°F., pulse 134, and respirations 24. He was coughing considerably, was very cyanotic and restless. He was placed in an oxygen tent immediately and this was continued up to the time of his death. Whenever it was necessary to continue the oxygen tent he became cyanotic and restless.

On the second postoperative day there was roentgenographic evidence of bronchopneumonia involving the right perihilar region and base of the right upper lobe. From this time on his temperature ranged from 103° to 105°F., the pulse averaged 130, and the respirations 30 per minute. He gradually grew weaker and expired the eighth postoperative day. At no time post-operatively was there any abdominal distention or pain.

Necropsy.—The stomach was slightly dilated, and the walls were thick and edematous. The duodenum was markedly dilated and, at a point approximately 3 cm. above the ligament of Treitz, there was a duodenojejunostomy. The stoma was of sufficient size and was apparently functioning well. There was no evidence of rupture or perforation at this site (Fig. 2A). Immediately distal to the stoma there was a shallow ulcer 1 cm. in diameter and approximately 1 cm. distal to this there was a completely annular ulcer, which was shallow and had thinned the jejunum at this site (Fig. 2B). The circumference of the jejunum at the ulcer was only 2.5 cm. The base and border of the ulcer was thick and fibrous. The pancreas, spleen, liver, and the remainder of the intestine were essentially normal.

Pathologic Examination—Microscopic: The stomach wall was thickened and edematous and the mucosa normal. In the jejunum there were a number of changes of considerable importance (Fig. 3). In the ulcer-bearing area, the wall was quite fibrous, and throughout the entire portion there was marked infiltration with lymphocytes, most of which were in definite collections. In this area, the plexus of Meissner as well as the ganglion fibers were distinctly swollen and edematous. The mucosal portion was distinctly hyperplastic and contained numerous mitotic figures. The area of hyperplasia merged imperceptibly with an area in which the epithelium was denuded and formed a definite ulcer. At this point there was a distinct penetration of the process into the muscular wall. The base was composed of dense fibrous connective tissue with numerous lymphocytes. There was some fibroblastic proliferation as evidenced by the attempt at repair. The glandular elements in the areas immediately adjacent to the ulcer were dilated and cystic. In the sections examined there was no evidence of heterotopic gastric mucosa.

Discussion.—In 1921, Judd[3] stated that not a single primary jejunal ulcer had been observed at the Mayo Clinic up to that time. Richardson[6] (1922) reported the first case in this country. Robinson and Wise[8] stated that only 13 cases of jejunal ulcer had been reported up to 1940, and most of these were from the European literature. They also stated that "in none of the case reports was a definite diagnosis made before operation." Ebeling[1] (1933) reported his case as the first on record to be diagnosed clinically, as the 42nd case reported in the literature, and the seventh case to be operated upon before perforation. Of the 42 cases, 35 were of the perforating variety and of these, 22 came to operation.

The etiology of primary nonspecific jejunal ulcer is as obscure as is that of the more common duodenal, pyloric, or gastric ulcer. When classified as nonspecific, it is significant that they are not due to any of the known etiologic agents, such as typhoid, dysentery, tuberculosis, syphilis, gastroenterostomy, trauma or tumors. The various theories as to the cause of these ulcers include focal infection, vascular obstruction and thrombosis, and hyperacidity from adjacent heterotopic gastric mucosa.

Most of the ulcers, in the cases reported, occurred in the proximal jejunum opposite the mesenteric attachment. With ulcer formation, stenosis occurs as a result of the connective tissue formed in and around the ulcer. Both macroscopically and microscopically the description is similar to that of chronic peptic ulcer. There are very few instances recorded of enlarged lymph nodes. Multiple ulcers were observed in nine of the cases reported.

These ulcers occur most frequently after the age of 20, the majority in middle life. They are twice as common in males as in females.

To accomplish a short-circuiting operation, it was necessary to divide the ligament of Treitz. This allowed mobilization of almost three inches of jejunum and duodenum. A lateral duodenojejunostomy around the obstruction was easily accomplished. Silk was used for the outer layer. Three finger tips could be inserted into the stoma. At the end of the operation the patient began to strain, vomited considerable bile, and became very cyanotic.

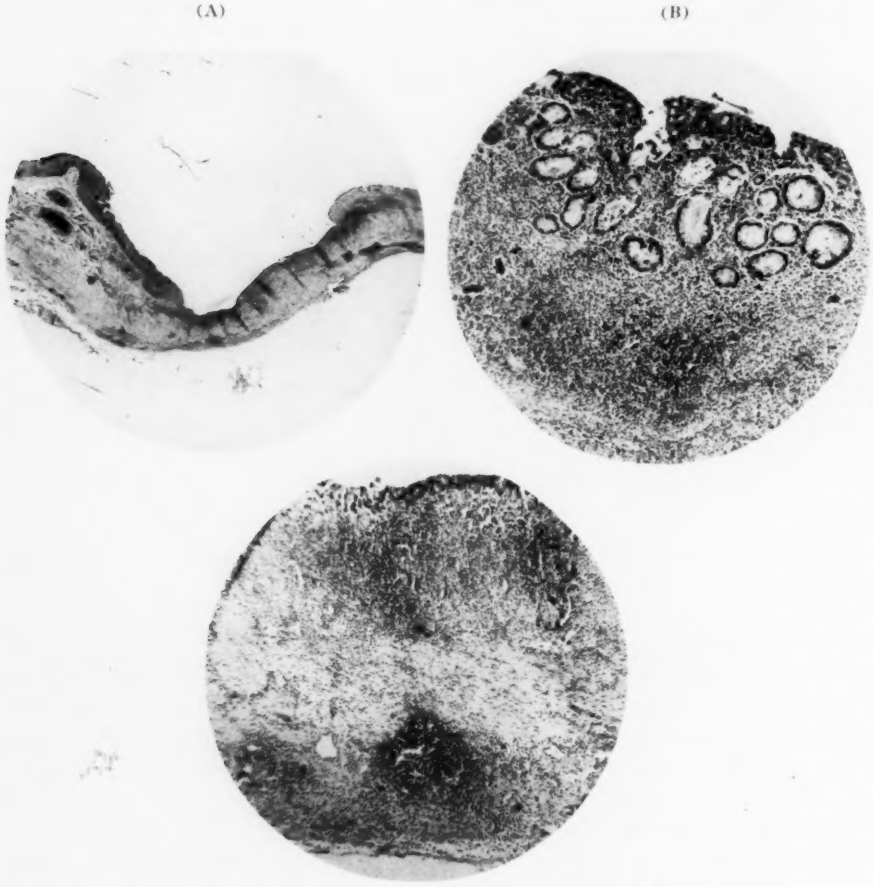


FIG. 3.—(A) Microscopic sections through the ulcer area—whole section through ulcer (Fig. 2). (B) The border of the ulcer. (C) The ulcer base.

Postoperative Course.—Within 24 hours following the operation the temperature was 106°F., pulse 134, and respirations 24. He was coughing considerably, was very cyanotic and restless. He was placed in an oxygen tent immediately and this was continued up to the time of his death. Whenever it was necessary to continue the oxygen tent he became cyanotic and restless.

On the second postoperative day there was roentgenographic evidence of bronchopneumonia involving the right perihilar region and base of the right upper lobe. From this time on his temperature ranged from 103° to 105°F., the pulse averaged 130, and the respirations 30 per minute. He gradually grew weaker and expired the eighth postoperative day. At no time post-operatively was there any abdominal distention or pain.

Necropsy.—The stomach was slightly dilated, and the walls were thick and edematous. The duodenum was markedly dilated and, at a point approximately 3 cm. above the ligament of Treitz, there was a duodenojejunostomy. The stoma was of sufficient size and was apparently functioning well. There was no evidence of rupture or perforation at this site (Fig. 2A). Immediately distal to the stoma there was a shallow ulcer 1 cm. in diameter and approximately 1 cm. distal to this there was a completely annular ulcer, which was shallow and had thinned the jejunum at this site (Fig. 2B). The circumference of the jejunum at the ulcer was only 2.5 cm. The base and border of the ulcer was thick and fibrous. The pancreas, spleen, liver, and the remainder of the intestine were essentially normal.

Pathologic Examination—Microscopic: The stomach wall was thickened and edematous and the mucosa normal. In the jejunum there were a number of changes of considerable importance (Fig. 3). In the ulcer-bearing area, the wall was quite fibrous, and throughout the entire portion there was marked infiltration with lymphocytes, most of which were in definite collections. In this area, the plexus of Meissner as well as the ganglion fibers were distinctly swollen and edematous. The mucosal portion was distinctly hyperplastic and contained numerous mitotic figures. The area of hyperplasia merged imperceptibly with an area in which the epithelium was denuded and formed a definite ulcer. At this point there was a distinct penetration of the process into the muscular wall. The base was composed of dense fibrous connective tissue with numerous lymphocytes. There was some fibroblastic proliferation as evidenced by the attempt at repair. The glandular elements in the areas immediately adjacent to the ulcer were dilated and cystic. In the sections examined there was no evidence of heterotopic gastric mucosa.

Discussion.—In 1921, Judd[5] stated that not a single primary jejunal ulcer had been observed at the Mayo Clinic up to that time. Richardson[6] (1922) reported the first case in this country. Robinson and Wise[8] stated that only 13 cases of jejunal ulcer had been reported up to 1940, and most of these were from the European literature. They also stated that "in none of the case reports was a definite diagnosis made before operation." Ebeling[1] (1933) reported his case as the first on record to be diagnosed clinically, as the 42nd case reported in the literature, and the seventh case to be operated upon before perforation. Of the 42 cases, 35 were of the perforating variety and of these, 22 came to operation.

The etiology of primary nonspecific jejunal ulcer is as obscure as is that of the more common duodenal, pyloric, or gastric ulcer. When classified as nonspecific, it is significant that they are not due to any of the known etiologic agents, such as typhoid, dysentery, tuberculosis, syphilis, gastroenterostomy, trauma or tumors. The various theories as to the cause of these ulcers include focal infection, vascular obstruction and thrombosis, and hyperacidity from adjacent heterotopic gastric mucosa.

Most of the ulcers, in the cases reported, occurred in the proximal jejunum opposite the mesenteric attachment. With ulcer formation, stenosis occurs as a result of the connective tissue formed in and around the ulcer. Both macroscopically and microscopically the description is similar to that of chronic peptic ulcer. There are very few instances recorded of enlarged lymph nodes. Multiple ulcers were observed in nine of the cases reported.

These ulcers occur most frequently after the age of 20, the majority in middle life. They are twice as common in males as in females.

The typical patient is a middle-aged male, who has a history of mild gastric distress, particularly pain in the epigastrium. This may simulate very closely the symptoms of gastric and duodenal ulcers, and with stenosis at the ulcer, there may be pain, distention and vomiting from obstruction. Melena may occur. The patient may have had no previous digestive complaints, but with perforation his symptoms will be similar to those of perforating gastric and duodenal ulcers.

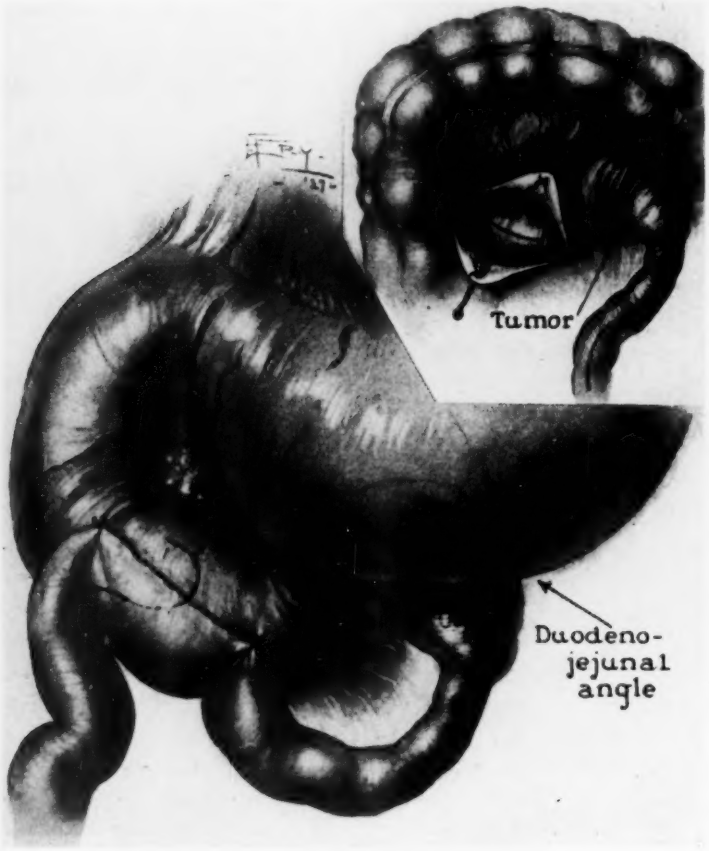


FIG. 4.—Drawing of proposed anastomotic method in obstructions near ligament of Treitz. (After Eusterman and Balfour[2]).

Clinically, the presence of simple nonspecific jejunal ulcer is hard to detect, as evidenced by the fact that only one was diagnosed up to 1934. Duodenal ileus should lead one to think of jejunal ulcer. Roentgenologic studies are invaluable in demonstrating these points.

Immediate operative repair is essential for the perforating ulcers. In the nonperforating types, either resection of the ulcer-bearing area or duodenojejunostomy may be performed. The usual duodenojejunostomy is

placed between the jejunum distal to the obstruction and the retroperitoneal duodenum to the right of the mesenteric vessels (Fig. 4).

In the present instance, the ligament of Treitz was cut and the duodenum was mobilized for a distance of about three inches above the point of obstruction, and was thus made available for anastomosis with the jejunum.

Dilatation of the duodenum, which was such an important factor in this patient, has been classified by Eusterman and Balfour[2] as follows: Chronic dilatation of the duodenum and chronic duodenal obstruction with dilatation. In the latter group the obstruction may be due to either intrinsic or extrinsic causes in relation to the bowel lumen.

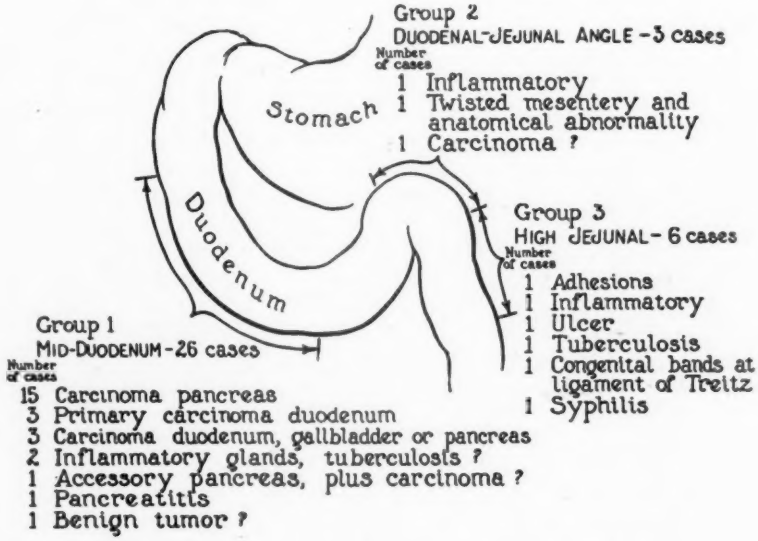


FIG. 5.—Causes of chronic duodenal obstruction with dilatation. (After Rivers and Thiessen[7]).

Among the intrinsic causes of duodenal obstruction are included the lesions of the duodenal wall producing stenosis and lesions or foreign bodies obstructing the lumen of the bowel from within. Among the extrinsic causes are included congenital anomalies and postoperative or inflammatory adhesions.

Rivers and Thiessen[7] reported 35 instances of chronic duodenal obstruction with dilatation, which were divided into three groups (Fig. 5).

In the intrinsic congenital types, septums within the bowel are the usual cause.

Of the intrinsic acquired causes of obstruction, carcinoma, lues, tuberculosis, jejunal ulcer, foreign bodies, gallstones, hairballs, and benign tumors are given as the causes.

The extrinsic types are much more frequent than the intrinsic, and the following are examples of this type: Congenital anomalies resulting in faulty rotation of the intestines; adhesive bands and membranes; postoperative adhesions and bands; extensive adhesions from chronic cholecystitis; growths

and tumors of neighboring organs and lymph nodes; and retroperitoneal new growths.

The diagonsis of chronic duodenal dilatation can not be made without roentgenologic examination. Krass and Beck[4] in an article on chronic duodenal ileus, stated that the diagnosis is exceedingly rare.

SUMMARY

The case report of a male, age 59, who had chronic dilatation of the duodenum produced by obstructive multiple jejunal ulcers, is presented. As a means of relief, a duodenojejunostomy was performed, in which the duodenum was mobilized by dissection after dividing the ligament of Treitz.

A brief review of pertinent factors pertaining to primary jejunal ulcer as well as duodenal dilatation due to obstruction is included.

BIBLIOGRAPHY

[1] Ebeling, Walter W.: Primary Jejunal Ulcer. ANNALS OF SURGERY, **47**, 857-874, June, 1933.

[2] Eusterman, G. B., and Balfour, D. C.: The Stomach and Duodenum. Philadelphia and London, W. B. Saunders Company, 1936, 382 pp.

[3] Judd, E. S.: Jejunal Ulcer. Surg., Gynec., & Obst., **33**, 120-126, August, 1921.

[4] Krass, E., and Beck, W. C.: Chronic Duodenal Ileus. ANNALS OF SURGERY, **99**, 311-331, February, 1934.

[5] Lahey, Frank H., and Marshall, Samuel F.: Some Unusual Gastro-enterological Problems, Am. J. Dig. Dis., **6**, 654-664, November, 1939.

[6] Richardson, E. P.: Jejunal Ulcer without Previous Gastro-enterostomy. Surg., Gynec., & Obst., **35**, 1-10, July, 1922.

[7] Rivers, A. B., and Thiessen, N. W. L.: Obstruction of the Upper Portion of the Small Intestine: A Clinical Study. Am. J. Dig. Dis., **1**, 92-96, April, 1934.

[8] Robinson, Walter D., and Wise: Simple Nonspecific Ulcers of the Jejuno-Ileum. Surg., Gynec., & Obst., **70**, 1097-1099, June, 1940.

MYO-EPITHELIAL HAMARTOMA OF THE ILEUM WITH INTUSSUSCEPTION

MARCUS E. COX, M.D.,

AND

EDWARD F. PARKER, M.D.

CHARLESTON, S. C.

FROM THE DEPARTMENTS OF PATHOLOGY AND SURGERY, MEDICAL COLLEGE OF SOUTH CAROLINA, CHARLESTON, S. C.

THE PURPOSE of this communication is to present an instance of an unusual benign tumor-like formation of developmental origin, composed of duct-forming epithelium and smooth muscle, occurring in the wall of the ileum of an infant, associated with intussusception; and to discuss briefly the general histologic structure, the classification, and the theories of origin of such a tumor.

The frequent association of tumors of the small intestine with intussusception is well recognized. Hence no further comment will be made on this association.

Benign tumors of supposedly developmental origin, composed of epithelium and smooth muscle in varying proportions and degrees of structural differentiation, such as aberrant, heterotopic, or incompletely differentiated accessory pancreatic tissue and adenomyomata, occurring in the gastro-enteric tract, are being reported in increasing numbers. However, until recently there were relatively few references in the literature to such tumors of the small intestine, with or without intussusception. In addition to numerous reports of polypoid adenoma, various other types of benign tumors of the small intestine have been reported, such as fibro-adenoma, leiomyoma, lipoma, fibroma, fibromyxoma, xanthoma, cystadenoma, hemangioma, lymphangioma, endometrioma, and dermoids.

King,[1] in 1917, in a review of 119 collected cases of benign tumors of the small intestine reported up to that time, did not mention the type to be described, nor did Willis,[2] Raiford,[3] Moore and Schmeisser,[4] Cave,[5] Joyce,[6] Fiske,[7] Goldberg,[8] or Cohn, Landy, and Richter.[9] Rankin and Newell,[10] in reporting 24 cases of benign tumor of the small intestine, described one case in which three adenomyomata were grouped together, causing partial obstruction. Clarke[11] has recently stated that tumor masses composed of smooth muscle and epithelium are occasionally encountered in the gastro-enteric tract and reported eight such cases along with pertinent references. He classified these tumors as hamartomata, using Albrecht's[12] term, with the additional descriptively qualifying term of "myo-epithelial" to indicate the type of tissue components.

Case Report.—Roper Hospital, No. 111,679: The patient, Negro, male, age nine months, was admitted to the hospital, July 23, 1940, suffering from constipation and

fever. Two days previously the infant had had its last bowel movement, which was said to have contained a few streaks of blood. On that day the infant vomited several times and was noted to be very fretful; the mother thought it also had abdominal pains because of frequent crying. The apparent pain persisted up until the time of admission. The following day the infant vomited four times and began to pass blood from the rectum. Thereafter there were frequent passages of blood. There was no history of any similar previous attack. As far as was known, the infant had been in good health since birth, without any previous serious illnesses.

Physical Examination.—Temperature 103°F., pulse 126, respirations, 28. The patient was a fairly well-nourished and well-developed Negro male baby who appeared acutely ill. There was no cyanosis or jaundice. The skin, lips, and tongue were dry. The mucous membranes were of good color. The heart and lungs were normal. The abdomen was diffusely distended, symmetrical and soft throughout. Over the upper abdomen there were visible patterns of distended loops of bowel. No mass was felt. The liver, spleen and kidneys were not palpable. There was no hernia. On continued observation, the abdominal wall intermittently became rigid, during which time the infant appeared to have abdominal pain. On rectal examination, no mass was palpable. After withdrawal of the examining finger, there was a bloody discharge from the rectum. The remainder of the physical examination was noncontributory.

W.B.C. 9,000 Hb. 8 Gm. Blood Wassermann and Kline negative. A specimen of urine was not obtained. Prior to operation a plain roentgenogram of the abdomen with the patient in the upright position showed moderate distention of the small intestine with numerous fluid levels present. Culture of the peritoneal fluid obtained at operation showed no growth.

The diagnosis of intussusception was made, and operation was performed immediately following the administration of parenteral fluids.

Operation.—Under drop-ether anesthesia, a right rectus, muscle-splitting incision was made. On incising the peritoneum, a small amount of clear, odorless yellow fluid escaped. A specimen for culture was obtained. Markedly distended loops of small bowel presented. An intussusception was found, with the head of the intussusceptum in the descending colon. Reduction was impossible without delivery, hence the intussusception was delivered, after which reduction was accomplished. The intussusception was ileocolic in type, starting about 30 cm. above the ileocecal valve. The terminal ileum was markedly discolored. However, later the color improved and the bowel appeared viable. At the site where the intussusception began, there was present in the wall of the ileum, on its antimesenteric border, a small, hard, round, tumor-like mass about one centimeter in diameter. There was puckering of the serosa over this mass. The mass was excised longitudinally and the defect closed transversely, leaving a satisfactory lumen in the bowel. There was no gross remnant of the omphalomesenteric duct. The abdomen was closed in layers without drainage, and the patient was returned to the ward in fair condition.

Following operation, the patient became dyspneic and cyanotic, the pulse rapid and weak, and the skin cold with perspiration. Death occurred about two and one-half hours later, in spite of supportive treatment. Blood donors could not be obtained. Permission for autopsy was not granted.

Pathologic Examination.—Gross: The surgical specimen consisted of a segment of small intestine, three centimeters in length, markedly discolored and apparently infarcted. The serosal surface was dull and covered by a thin layer of fibrin; the wall was generally dusky and succulent, and the mucosal surface deeply discolored. There was a bulbous thickening between the mucosa and serosa which on section showed a small tumefaction of muscular appearance, measuring 1.3 cm. in its greatest diameter. It did not appear to be sharply outlined. *Microscopic:* Sections taken through the nodule showed it to be composed of single and multiple groups of closely arranged duct-like and cystic spaces surrounded by irregularly arranged interlacing bands of smooth muscle. Edematous

fibrous tissue supported and engulfed the smooth muscle and epithelial-lined spaces and accounted for the considerable bulk of the tumor (Fig. 1). About the periphery of the larger cyst-like spaces of bizarre outline, presenting intracystic papilliferous projections which tended to coalesce, were smaller, isolated duct-like spaces surrounded by smooth muscle. Smooth muscle formed the basement layer for the epithelium of the duct type in most instances. It did not appear to be an intrinsic part of the muscualture of the intestinal wall, but possibly derived from the latter. The epithelium lining the duct structures was of the tall columnar undifferentiated type with eosinophilic cytoplasm and prominent basal nuclei, not unlike that of pancreatic duct epithelium in its appearance (Fig. 2). In some instances the epithelium was crowded into a pseudostratified

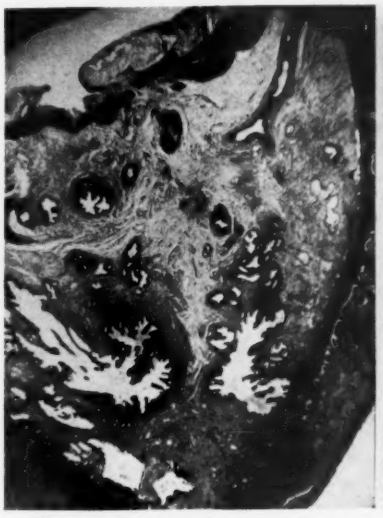


FIG. 1.—Low power view of tumor showing the epithelial lined cyst-like and duct-like spaces surrounded by smooth muscle supported by edematous fibrous tissue.

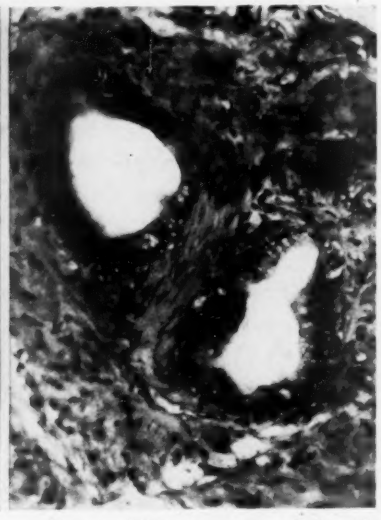


FIG. 2.—High power view of duct-like spaces lined by tall columnar epithelium not unlike that of pancreatic duct epithelium.

arrangement. At the bases of some papillary projections there were noted acinar-like structures whose lining cells showed clear cytoplasm and small compressed basal nuclei, suggesting an attempted formation of the Brunner-type gland, though this feature was not conspicuous. The epithelial-lined ducts and cystic spaces were observed to be immediately subjacent to the mucosa in some areas but communication of the latter with the lumen of the intestine was not demonstrated. The overlying mucosa showed necrosis, leukocytic infiltration and heavy surface exudation.

DISCUSSION.—Tumors of developmental origin occurring along the gastroenteric tract are most frequently described as occurring in the gastroduodenal region where mishaps in the complicated process of development are most apt to occur. They occur to a lesser extent, though rather commonly, along the lower intestine, particularly in the ileum and jejunum. The essential component tissues of such tumors are epithelium and smooth muscle, though the latter has not been emphasized to the same degree as the former. Most often the epithelial elements show varying degrees of structural differentiation, commonly toward pancreatic tissue, with or without duct-like structures lined by undifferentiated columnar epithelium. Hence, the most common category

into which these tumors have been placed is aberrant or heterotopic pancreatic tissue. Islet tissue and Brunner-type glands have been reported as associated features in some cases. In those instances in which duct-like structures are the most conspicuous and the glandular differentiation less pronounced, or lacking, Lauche[13] has suggested the term, "incompletely differentiated accessory pancreas." When duct-like structures usually lined by a single layer of undifferentiated columnar epithelium, in association with closely related smooth muscle, are found, the tendency has been to use the designation of adenomyoma,[14, 15] indicative of neoplasia and the types of component tissues, though the ability of these tumors to grow has not been definitely proved.

It has been the tendency on the part of those interested in the explanation of the origin of such tumors to regard them as heterotopias of developmental origin, derived from misplaced epithelial buds or diverticula during the process of development of the intestinal tract. Several theories of origin of aberrant pancreatic tissue in the stomach and duodenum have been advanced. Zenker[16] believed them to be anomalies of early life on the basis of an extra diverticulum from the duodenum. Glinski[17] hypothesized failure of coalescence of one or more anlages of the pancreas in cases of accessory pancreatic tissue in the duodenum. Warthin[18] believed that lateral budding of the rudimentary pancreatic ducts occurred as they passed through the duodenal wall. Obviously these theories do not explain the occurrence of such aberrant tissue in remote sites such as the lower intestinal tract. Horgan[19] suggested the possibility that branching buds of the pancreatic anlage came into contact with remote sites and became engrafted. Lauche[13] reserved the term adenomyoma for heterotopias whose differentiation was not toward the pancreatic tissue type. King and MacCallum[20] explained the genesis of accessory pancreatic tissue in the stomach wall on the basis of a post-inflammatory sequela, following a study of four cases, all over 45 years of age, which cases presented demonstrable associated chronic inflammatory changes, apparent development by differentiation from gastric mucosa under inflammatory stimuli and direct connection of the glandular structure with atypical gastric mucosa. King and MacCallum[20] believed that the close association of the glands with smooth muscle and the failure to demonstrate any relation of the associated duct-like structures with the surface were factors against the cell rest theory of Cohnheim. Clarke[11] advocated that such localized tumors of the gastro-enteric tract of epithelial and smooth muscle composition, regardless of their relative proportions and the degree of structural differentiation, be regarded as myo-epithelial hamartomata. He saw no purpose in endeavoring to classify such occurrences, manifesting such broad structural differentiation, as to the type of tissue most closely simulated in the particular instance of this group. He believed that the mechanism of formation was on the basis of misplaced epithelium occurring during the stage of embryonal development of the tract, and that the smooth muscle was independent of the muscle of the intestinal wall but possibly derived from the latter in response to stimuli provided by misplaced epithelium.

The variation in the structural arrangement of the epithelium, ranging from undifferentiated duct type epithelium lining spaces to more specialized glandular formation embracing pancreatic and Brunner's-type acini or mixtures of these elements should be regarded as reflecting degree of differentiation, rather than dissimilar origin.

We are in accord with Clarke's viewpoint and believe that the tumor in this case is best classified as a myo-epithelial hamartoma.

SUMMARY

1. A tumor-like formation composed of epithelial-lined ducts and cystic spaces in intimate association with smooth muscle, of developmental origin, associated with intussusception in an infant, is reported.

2. Justification for classification of the lesion as a myo-epithelial hamartoma is offered.

REFERENCES

[1] King, E. L. Benign Tumors of the Intestines: With Special Reference to Fibroma. Surg., Gyn. and Obs., **25,** 54, 1917.

[2] Willis, A. M.: Intussusception Resulting from Benign Tumor of the Intestine. Surg. Gyn. and Obs., **30,** 603, 1920.

[3] Raiford, T. S.: Tumors of the Small Intestine. Arch. Surg., **25,** 122, 1932.

[4] Moore, R. M., and Schmeisser, H. C.: Benign Tumors of the Small Intestine. South. Med. Jour., **27,** 386, 1934.

[5] Cave, H. W.: Tumors of the Small Intestine. ANNALS OF SURGERY, **96,** 269, 1932.

[6] Joyce, T. M.: Tumors of the Small Intestine. ANNALS OF SURGERY, **100,** 949, 1934.

[7] Fiske, F. A.: Intussusception due to Intestinal Tumors. ANNALS OF SURGERY, **105,** 221, 1937.

[8] Goldberg, S. A.: Unusual Neoplasms of the Small Intestines. Am. Jour. Clin. Path., **9,** 516, 1939.

[9] Cohn, S., Landy, J. A., and Richter, M.: Tumors of the Small Intestine. Arch. Surg. **39,** 647, 1939.

[10] Rankin, F. W., and Newell, C. E.: Benign Tumors of the Small Intestine. Surg., Gyn. and Obs., **57,** 501, 1933.

[11] Clarke, B. E.: Myoepithelial Hamartoma of the Gastro-Intestinal Tract. Arch. Path. **30,** 143, 1940.

[12] Albrecht, E.: Quoted by Mallory, Tracy, B.: New Eng. Jour. Med., **218,** 1105, 1938; and by Goldsworthy, N. E., Jour. Path. and Bact., **39,** 291, 1934.

[13] Lauche, A.: Die Heterotopien des Ortsgehörigen Epithels im Bereich des Verdauungskanals. Virchow's Arch. f. path. Anat., **252,** 39, 1924.

[14] Woolsey, J. H., and Miltzner, R. J.: Adenomyoma of the Stomach. Arch. Surg., **16,** 583, 1928.

[15] Stewart, M. J., and Taylor, A. L., Adenomyoma of the Stomach. Jour. Path. and Bact., **28,** 195, 1925.

[16] Zenker, F. A.: Nebenpankreas in der Magenwand. Virchow's Arch. f. path. Anat., **21,** 369, 1861.

[17] Glinski, L. K.: Zur Kenntnis des Nebenpankreas und verwandter Zustände. Virchow's Arch. f. path. Anat., **164,** 132, 1901.

[18] Warthin, A. S.: Two Cases of Accessory Pancreas. Physician and Surgeon, **26,** 337, 1904.

[19] Horgan, E. J.: Accessory Pancreatic Tissue: Report of Two Cases. Arch. Surg., **2,** 521, 1921.

[20] King, E. S. J., and MacCallum, P.: Pancreatic Tissue in the Wall of the Stomach. Arch. Surg., **28,** 125, 1934.

A STUDY OF THE BACTERIOLOGY OF THE COMMON BILE DUCT IN COMPARISON WITH THE OTHER EXTRAHEPATIC SEGMENTS OF THE BILIARY TRACT

GERARDO ELKELES, M.D.,

AND

PROF. P. L. MIRIZZI, M.D.

CORDOBA, ARGENTINA

PREVIOUS STUDIES of the bacteriology of the biliary tract have been restricted to investigations of the wall of the gallbladder and its contents, the regional nodes, and the duodenum. We can find no systematic study in the literature of the bacteriology of this region which has included the common bile duct of the same patients in whom the other portions were studied.

The following technic was employed in obtaining material for this study. Common duct bile was removed through the wall of the duct only in cases in which choledochotomy was to be performed. In the majority of cases it was obtained by introducing a cannula with an olive-shaped tip through the stump of the cystic duct. One or two cubic centimeter-specimens of common duct bile were then aspirated. This procedure has the advantage that it eliminates the possibility of damaging the wall of the common duct. However, it has the disadvantage of contaminating the common duct bile from the cystic duct. That this actually occurred in exceptional instances will be referred to later (Series III). As such contamination occurred rarely, we persisted in the use of this technic. In those instances in which the valves of Heister offered marked resistance to the passage of the cannula, the attempt to pass it was given up, in accordance with the principle of *primum non nocere*. For this reason, specimens of common duct bile were obtained in only 75 of 138 cases studied.

In addition to the common duct bile, specimens were obtained from the gallbladder and from the duodenum in all cases, as was a piece of the wall of the gallbladder. Whenever a sediment was obtained from the material, stained microscopic smears were prepared and cultures made on agar and in broth. Large quantities of broth were used in order to prevent any possible bactericidal action by the substrate on the bacteria being sought. The specimens from the wall of the gallbladder were taken from the fundus, passed through alcohol, and flamed several times. They were then triturated and placed in broth. Care was taken to prevent contamination. Aerobic gram-positive spore-forming bacteria frequently grew out. As yet, it has not been possible to judge exactly the origin and importance of these bacteria. In general, they did not appear in cultures of the gallbladder wall containing pathogenic bacteria, such as *B. coli,* streptococcus, and *B. typhi.* For the present, we are assuming that these organisms were contaminants.

The cystic lymph node was examined in several cases. In most instances it proved to be sterile and for this reason routine study of the node was discontinued.

All cultures were incubated for three days. The more extensive bacteriologic technics were not employed because the object of the study was not to gain more knowledge of the bacteria causing inflammation of the biliary tract, but rather to make a comparative bacteriologic study of the extrahepatic segments of the biliary tract, and particularly of the bacteriology of the common bile duct in comparison with that of the other segments. Table I gives a general orientation on the results obtained from the bacteriologic studies. The flora of the common bile duct, in comparison with that of the other segments, is emphasized in the table by the numerical occurrence of the different possibilities. The significance of the relationships shown in the table are then discussed in the same order in which they are presented.

Series I.—In this group, not only was the culture of the common duct bile negative, but also the cultures of the gallbladder bile, the gallbladder wall, and the duodenal contents, were sterile. There was 25 such cases. Twenty of these cases were instances of uncomplicated chronic calculous cholecystitis, atrophic sclerosing cholecystitis, or hydrops of the gallbladder. Three were cases of uncomplicated hydrops of the gallbladder, with stones. One case (No. 88) had a calculus lodged in the papilla of Vater, in addition to a chronic calculous cholecystitis, and the remaining case was that of a patient who was operated upon for adhesions following a previous cholecystectomy. It is evident, therefore, that 23 of the 25 cases of general sterility are cases of uncomplicated gallbladder disease, such as chronic calculous cholecystitis, or hydrops of the gallbladder. In Case No. 88, in which there was a calculus in the papilla of Vater, the common duct bile was sterile and of normal appearance, as were the other samples. The cases comprising Series IV show that in all cases of common duct stone there is bacterial infection of the common duct bile, with some change in its appearance. The fact that there was no infection of the choledocal bile in Case No. 88 seems to indicate that the passage of the calculus from the gallbladder toward the duodenum had occurred so recently that functional or organic alterations had not as yet been produced by the calculus.

TABLE I

COMPARATIVE RESULTS OF BACTERIOLOGIC EXAMINATIONS OF BILE FROM THE COMMON BILE DUCT, WALL AND CONTENTS OF THE GALLBLADDER AND DUODENAL JUICE—TOTAL NUMBER OF CASES EXAMINED—75

Choledocal Bile *without* Bacteria—41 cases		Choledocal Bile *with* Bacteria—34 cases		
The other cultures (—)—25	The other cultures (+)—16	The other cultures (—)—0	The other cultures (+)—34	
			Without lithiasis of the common bile duct—18	With lithiasis of the common bile duct—16
Reference in course of the article				
Series I	Series II		Series III	Series IV

Key: Sign minus (—) = *without* bacteria.
Sign plus (+) = *with* bacteria.

Series II.—In the cases in this series the common duct bile was sterile, whereas the other samples, namely, the wall and bile of the gallbladder and the duodenal juice, were contaminated by bacteria. There were 16 such cases, which are described in detail in Table II. Before analyzing the data in this

table, it is advisable to make a rough comparison of the data in Series II, III, and IV as they appear in Tables II, III, and IV. When this is done, a remarkable irregularity of the bacteriologic findings in Table II will be noted. If the two cases of choledocal lithiasis (Nos. 45 and 32) are excluded, there remain only two cases (Nos. 8 and 111) in which the bacterial flora was the same in the three different specimens (gallbladder bile, gallbladder wall, and duodenal juice). In contrast to this, the flora was the same in most of the

TABLE II

CHOLEDOCAL BILE (—). CULTURES OF OTHER STRUCTURES OF THE BILIARY SYSTEM (+)—TOTAL NUMBER OF CASES 16

SERIES II

No. of the Case	Operative Diagnosis	W	G	D	Macroscopic Alterations of Choledocal Bile
56	Chronic calculous cholecystitis	Cn.	—	Enterococ. & *Staphylo. alb.*	○
84	Chronic calculous cholecystitis	Cn.	Agar broth. Cn.	Agar broth. Enterococ. and yeasts	○
85	Chronic calculous cholecystitis	Cn.	Cn.	Associated bact.	○
122	Chronic calculous cholecystitis	Cn.	*B. pyocyaneus*	./.	○
111	Sclerosing, atrophic, calculous cholecystitis	Coliform bact.	Coliform bact.	Coliform bact.	◉
8	Sclerosing, atrophic, calculous cholecystitis Cysticoduodenal fistula	Associated bact. flora	Associated bact. flora	Associated bact. flora	○
87	Suppurative cholecystitis	— Cn.	Enterococ.	*B. coli* *B. coli* and	◉
14	Calculous empyema of gallbladder	Staphylococ.	—	staphylococ.	○
A	Calculous empyema of gallbladder	Cn. Enterococ.	—	—	./.
50	Hydrops of calculous gallbladder; pericholecystitis and chronic appendicitis	Enterococ.	—	—	◉
37	Hydrops of calculous gallbladder and appendicular peritonitis	*B. coli*	—	—	○
30	Chronic calculous cholecystitis and dyskinesia	—	Gram-pos. cocci. Cn?	Staphylococ. and Sarc.	●
20	Chronic calculous cholecystitis; stenosing odditis and angiocholitis?	Cn.Streptococ.	—	./.	●
36	Stenosing odditis; pancreatic stricture	./.	./.	Staphylococ. Enterococ.	○
45	Chronic calculous cholecystitis and choledocal lithiasis	*B. coli*	*B. coli*	*B. coli*	○
32	Calculous empyema of gallbladder; solitary secondary choledocal lithiasis	*B. coli*	*B. coli*	*B. coli*	○

Key: Cn. = contamination; ./. = not examined; (—) = without bacteria; (+) = with bacteria; ○ = without macroscopic alteration; ◉ = slight macroscopic alterations; ● = acute macroscopic alterations; W = wall of gallbladder; G = bile of gallbladder; C = choledocal bile; and D = juice of duodenum.

cases in Series III and IV. Furthermore, in the cases in Series II the bacteria of moderate virulence were predominant and bacterial contamination was frequent. It is quite probable that many of the bacteria identified were not the active causal agent of the disease and that these cases really belonged in the first series in which the bile of the common duct and of the other parts of the extrahepatic biliary system was sterile. In three cases (Nos. 20, 30 and 36) the choledocal bile was sterile in the presence of stenosing odditis or dyskinesia which were verified by cholangiography during operation. Thus, it seems that bile stagnation does not necessarily give rise to infection of the choledocal bile, not even in cases where microscopic alterations

of the bile exist (Nos. 20 and 30). In this series there were two cases (Nos. 32 and 45) in which the choledocal bile was sterile and of normal appearance in the presence of calculi. It is probable that the sterility of the choledocal bile in these two cases could be explained in the same way as in Case No. 88 in Series I. The fact that the common duct bile was sterile in these cases in which cultures of the wall and bile of the gallbladder and of the duodenal

TABLE III

CHOLEDOCAL BILE (+). CULTURES OF OTHER STRUCTURES OF THE BILIARY SYSTEM (+). COMMON BILE DUCT WITHOUT CALCULI. Total number of cases 18.

SERIES III

No. of the Case	Operative Diagnosis	W	G	C	D	Macroscopic Alterations of Choledocal Bile
16	Chronic calculous cholecystitis	*B. proteus*	*B. proteus*	*B. proteus*	*B. proteus*	◉
19	Chronic calculous cholecystitis	*B. coli*	*B. coli*	*B. coli*	*B. coli*	◉
27	Chronic calculous cholecystitis	*B. typhi*	*B. typhi*	2 colonies of *B. typhi*	*B. typhi*	○
100	Chronic calculous cholecystitis	Enterococ.	Enterococ.	Agar broth. Enterococ.	Enterococ.	○
108	Chronic calculous cholecystitis	*B. coli*	*B. coli*	2 colonies of *B. coli*	*B. coli*	○
116	Chronic calculous cholecystitis	Streptococ.	Streptococ.	3 colonies of streptococ.	Streptococ.	○
113	Sclerosing, atrophic calculous cholecystitis	Cn.	*B. coli*	Agar broth. *B. coli*	*B. coli*	○
129	Sclerosing, atrophic calculous cholecystitis	Cn. Enterococ.	Enterococ.	2 colonies of enterococ.	Enterococ.	◉
72	Sclerosing, atrophic calculous cholecystitis, Empyema	*B. pyocyaneous*	*B. pyocyaneous*	Isolated colonies of *B. pyocyaneous*	*B. pyocyaneous*	○
79	Chronic cholecystitis, without concretions	*B. coli*	*B. coli*	*B. coli* (small number)	*B. coli*	◉
66	Chronic calculous cholecystitis and pericholecystitis	*B. typhi*	*B. typhi*	*B. typhi*	*B. typhi*	◉
11	Chronic cholecystitis, without concretions and pericholecystitis	Cn.	Enterococ.	Enterococ.	Enterococ.	●
114	Chronic cholecystitis, without concretions and pericholecystitis	*B. coli*	*B. coli*	*B. coli*	*B. coli*	●
120	Sclerosing, atrophic cholecystitis, without concretions, hepatitis and appendicitis	./.	Agar broth. *B. coli*	*B. coli*	./.	●
102	Chronic calculous cholecystitis; dyskinesia	Staphylococ.	Streptococ. *B. coli*	*B. coli*	*B. coli*	○
92	Chronic calculous cholecystitis; stenosing odditis	./.	./.	Enterococ.	./.	○
63	Sclerosing, atrophic cholecystitis, without concretions and stenosing pancreatitis	Enterococ.	Enterococ.	Enterococ.	Enterococ.	◉
73	Hydrops of calculous gallbladder and stenosing pancreatitis	Staphylococ.	—	Agar broth. Staphylococ.	Agar broth. Staphylococ.	◉

Key: See those appended to Table II.

juice contained pathogenic bacteria of identical species tends to show the strong bactericidal power of the choledocal bile, which may be sterile in the presence of infection in all the other extrahepatic segments of the biliary tract.

Series III.—The 18 cases in this series yielded specimens which were infected in every instance. In none of the cases, however, were there stones in the common duct. The uniformity of the bacteriologic results in the different parts of the biliary system in the third and fourth series is sig-

nificant and tends to verify the etiologic rôle of the identified bacteria in the development of the pathologic process. There are, however, certain quantitative bacterial differences between the choledocal bile and the other specimens which do not exist in the cases of the first and second series. They are of significance especially because they were seen only in uncomplicated processes in the gallbladder, generally associated with sterility of the choledocal bile. Cases 27, 100, 108, 113, 116 and 129 are of particular interest in this regard and Cases 72 and 79 may also be considered. A heavy layer of bacteria grew on agar cultures of the wall and bile of the gallbladder and of the duodenal juice, whereas only isolated colonies of bacteria developed in cultures of the choledocal bile prepared with the same amount of material as that used in the other cultures. We believe that contamination of the choledocal bile, rather than actual infection would give a plausible explanation for the scantiness of the growth in these cultures. The growth probably occurred because some of the large number of bacteria which were present in other parts of the biliary system were introduced into the common duct bile at the time the cannula was passed through the stump of the cystic duct. Further support is given to this interpretation by the fact that the choledocal bile had a normal microscopic appearance in six of the eight cases of this nature in Series III, whereas it was more or less altered in six of the eight complicated cases (Nos. 63, 66, 73, 102, 114 and 120) in the same series. There was organic or functional stagnation in four of these cases (Nos. 63, 73, 92 and 102). These cases were complicated by dyskinesia, stenosing odditis and stenosing pancreatitis. Infection was present in these cases, and the macroscopic appearance of the common duct bile was normal in Cases 92 and 102 and altered in Cases 63 and 72.

Series IV.—This series comprised 16 cases with infection of the common duct bile, accompanied by common duct stones. The wall and bile of the gallbladder and the duodenal juice were infected. Details are presented in Table IV. The bile of the common duct was infected in all cases with choledocal lithiasis, except Case No. 88 of the first series, in which the calculus was lodged in the papilla of Vater and in Cases No. 32 and 45 of the second series. In the 16 cases in the fourth series with common duct stones, the bacteria were of the same type as that identified in the wall and bile of the gallbladder and in the duodenal juice. This is taken as evidence that the bacteria recovered had an etiologic rôle in the development of the infection. The clinical records show that the macroscopic appearance of the common duct bile was more or less altered in all the cases of this series.

Previous investigations, by various authors, have shown that suppuration within the gallbladder frequently results in autosterilization of the gallbladder contents. This may occur especially in cases of inflammatory edema at the neck of the gallbladder, which causes occlusion. The fact that bacterial infection of the vascular bile existed previously can often be concluded from stained preparations, which show bacterial rests within a large number of cells. Even when the stained preparations show bacteria with well preserved

TABLE IV

CHOLEDOCAL BILE (+). CULTURES OF OTHER STRUCTURES OF BILE SYSTEM (+). CALCULI IN THE COMMON BILE DUCT. Total number of cases 16.

SERIES IV

No. of the Case	Operative Diagnosis	W	G	C	D	Macroscopic Alterations of Choledocal Bile
133	Chronic calculous cholecystitis; choledocal lithiasis	Cn.	./.	*B. coli* Enterococ. Saprophyt. bact.	./.	●
95	Chronic calculous cholecystitis; choledocal lithiasis	./.	./.	*B. coli*	./.	◉
22	Chronic calculous cholecystitis; multiple secondary lithiasis	*B. coli* Enterococ.	*B. coli* Enterococ.	*B. coli* Enterococ.	*B. coli* Enterococ.	◉
52	Sclerosing, atrophic, chronic calculous cholecystitis; solitary choledocal lithiasis; chronic appendicitis	Enterococ.	Enterococ.	Agar broth. Enterococ.	—	◉
13	Sclerosing, atrophic, chronic calculous cholecystitis; multiple choledocal lithiasis	./.	*B. coli*	*B. coli*	*B. coli*	◉
77	Sclerosing, atrophic, chronic cholecystitis, without concretions; solitary choledocal lithiasis	./.	Agar broth. 48 hours. *B. coli*	*B. coli*	./.	●
51	Sclerosing, atrophic cholecystitis; multiple choledocal lithiasis	./.	*B. coli*	*B. coli*	*B. coli*	●
128	Chronic calculous cholecystitis; multiple secondary choledocal lithiasis	*B. coli*	*B. coli*	*B. coli*	*B. coli*	◉
93	Chronic calculous cholecystitis; choledocal lithiasis; stenosing odditis	*B. coli*	*B. coli*	Isolated colonies of *B. coli*	*B. coli*	◉
101	Chronic calculous cholecystitis; choledocal lithiasis; stenosing odditis	Cn.	*B. coli*	*B. coli*	*B. coli*	./.
90	Chronic calculous cholecystitis; choledocal lithiasis; stenosing pancreatitis	./.	Isloated colonies of *B. coli*	*B. coli*	*B. coli*	●
130	Chronic calculous cholecystitis; choledocal lithiasis; stenosing pancreatitis	./.	./.	Streptococ.	./.	●
48	Chronic calculous cholecystitis; multiple secondary choledocal lithiasis; stenosing odditis	*B. coli* Enterococ.	*B. coli* Enterococ.	*B. coli* Enterococ.	*B. coli* Enterococ.	●
39	Perforated calculous cholecystitis; secondary choledocal lithiasis	Enterococ. Staphylococ.	—	Enterococ.	Isolated colonies of enterococ.	◉
81	Chronic calculous cholecystitis; solitary calculous of hepatic duct	*B. coli* Enterococ.	*B. coli* Enterococ.	*B. coli* Enterococ.	*B. coli* Enterococ.	◉
57	Multiple choledocal lithiasis with recurrences after cholecystectomy and choledocotomy	./.	./.	*B. coli* Enterococ.	*B. coli* Enterococ.	●

Key: See those appended to Table II.

staining properties, the cultures frequently remain sterile, whereas in other parts of the biliary system, bacteria of the same type may be recovered. Illustrations of this type of autosterilization, either partial or complete, occurred in Cases No. 39, 77, and 90 of the fourth series, in Cases No. 73, and 120 of the third series, and in Cases No. 14 and 20 in the second series.

Quantitative differences between bacteria in the common bile duct and in other parts of the biliary system were observed infrequently in the presence of choledocal calculi (Cases No. 52 and 93).

CONCLUSIONS

1. Choledocal bile is sterile in a large number of patients with disease of the extrahepatic bile ducts. In our operative material this occurred in 41 of 75 cases, or 54.7 per cent. If a correction is made for cases in which bacterial contamination was probable, the figure would be 64 per cent.

2. Sterility of the choledocal bile is especially frequent in cases of uncomplicated chronic calculous cholecystitis and atrophic sclerosing cholecystitis and was observed in 25 to 34 such cases, or 73.5 per cent. Since the possibility of contamination could not be excluded in seven of the ten remaining cases of uncomplicated cholecystitis in the third series, the frequency of sterile common duct bile in such cases may be about 91 per cent. The results were similar in cases of empyema, or hydrops of the gallbladder, when the gallbladder was the only organ involved by the disease. On the other hand, the choledocal bile was frequently infected when there were complications, either of an organic nature, such as pericholecystitis, hepatitis, odditis, or of a functional nature, such as dyskinesia.

3. Stagnation of the choledocal bile, resulting from dyskinesia, or stenosing odditis, in the absence of stone, resulted in infection of the common duct bile in four of seven cases. In the remaining three cases actual infection was not observed, although macroscopic alterations of the choledocal bile existed.

4. Stones in the common duct were the most frequent cause of infection of the choledocal bile. Infection occurred in 16 of 18 cases of choledocal lithiasis, or 88.8 per cent. In the two remaining cases, the choledocal bile did not contain bacteria and had a normal macroscopic appearance. It seemed probable that in these two cases the calculus found during the operation in the common duct was migrating from the gallbladder to the duodenum. Probably, it is only under these circumstances that the choledocal bile may be sterile in the presence of common duct stones. The predominant bacteria in the various parts of the biliary system were *B. coli,* which occurred 72 times, and streptococcus, which occurred 49 times. Other bacteria recovered were *B. typhi* (eight times), *B. pyocyaneus* (four times), and staphylococcus, saprophytes, and associated bacteria (13 times).

5. Bacterial infection of the common duct is always associated with infection of the other parts of the biliary system and the bacteria found are generally of the same species.

6. The choledocal bile appears to have strong bactericidal power. This finding is striking and of great practical importance.

We hope that similar bacteriologic studies will be undertaken in other clinics.

The operative material was obtained in the Clinical Ward of the Surgical Clinic, of the Faculty of Medicine, of the Universidad Nacional de Cordoba, Argentina, of which Prof. Pablo L. Mirizzi is the head. The bacteriologic researches were made in the Bacteriologic Laboratory, of the Chair of Pediatrics, of Prof. Jose M. Valdes, of which the author is the head.

The details of the technic appear on page 177 of Vol. 5 of Actas y trabajos of the Sixth National Congress of Medicine, which was held in Cordoba, October 16-21, 1938.

ACUTE PANCREATITIS

REPORT OF TWENTY-NINE CASES

R. STARR LAMPSON, M.D.

HARTFORD, CONN.

FROM THE HARTFORD HOSPITAL, HARTFORD, CONN.

ACUTE PANCREATITIS is a bizarre disease difficult to diagnose, and easy to confuse with any other of the many acute intra-abdominal conditions. In its severest form it is a serious and not infrequently fatal ailment. The first accurate description and presentation of acute pancreatitis as a clinical entity was given by Reginald Fitz,[1] in 1889, when he published a report of 15 cases. Only two of these patients recovered. Fitz clearly described the hemorrhagic destruction of the pancreas and the associated fat necrosis, and felt that it was due to a gastro-enteritis extending up the pancreatic duct. Opie[2] demonstrated, in 1901, that retrograde injection of the pancreatic duct with bile produced a fatal pancreatitis in dogs. An important contribution was made by Wohlgemuth,[3] in 1910, when he reported that in irritative conditions of the pancreas, the pancreatic ferments can be demonstrated in increased amounts in the urine and blood. In 1934, Mikkelsen[4] presented a series of cases, with a mortality of 7.5 per cent by the conservative treatment of pancreatitis. This was such a low mortality as compared with the then recognized mortality of 40 to 50 per cent[5] that his figures were looked upon with skepticism. Mikkelsen used the diastase test as the main criterion for diagnosis and, therefore, included milder cases in which the diagnosis otherwise might never have been made. Since this stimulating work, several papers[6, 7, 8, 9, 10, 11] have been published which tend to confirm his contentions and to substantiate his principles of conservative management of patients with acute pancreatitis.

The data which is herewith presented represents the results of our study of this subject on the Surgical Services and in the Hall Wilson Laboratory of the Hartford Hospital, from September 1, 1938, to December 31, 1941.

MATERIAL.—The clinical material has been taken from the private and ward surgical services, and includes patients in whom the diagnosis has been made by operation, autopsy, or clinical evidence associated with an elevated urinary diastase. We have used the method of diastase determination described by Foged[12] and have found it very satisfactory. Foged's diastase determination depends upon the hydrolization of starch by measured dilutions of urine. The mixture is incubated and then iodine is added to determine the starch free tubes. The diastase activity is expressed in units of which one unit is arbitrarily taken to be the amount of diastase necessary to hydrolize 1 cc. of 0.1 per cent starch solution under standard conditions. Normal urine shows a diastase activity of less than 300 units. A finding above this

is abnormal and indicates an acute irritative pancreatic process. We have observed no proven false positives as every case operated upon in the acute stage of the disease, on whom the test has been made early, has shown an elevated diastase. It should be emphasized that the test must be made early in the disease, and fresh urine should be used (Chart I) as the activity of

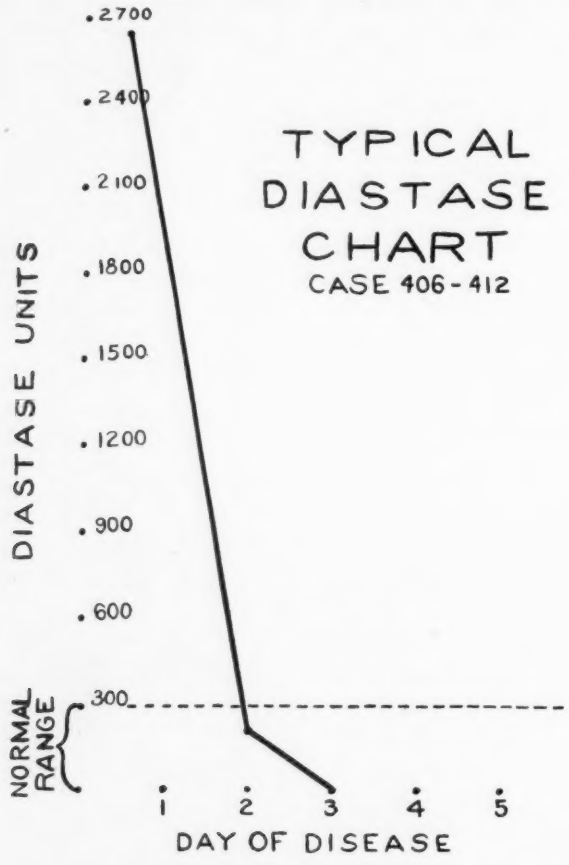


CHART 1.

the enzyme deteriorates on standing. We have used the urine rather than the blood because it is simpler to obtain, requires less manipulation in the laboratory, and we feel gives accurate results.

Symptoms and Findings.—In a little over three years, there were 29 patients seen in the Hartford Hospital with acute pancreatitis. The severity of the illness varied from a rather mild abdominal upset to an overwhelming and sometimes fatal illness. The onset was usually abrupt with upper abdominal pain accompanied by nausea with vomiting. The pain often radiated to the back. Jaundice sometimes was present. The physical findings varied in degree but consisted chiefly of upper abdominal tenderness and spasm. High epigastric tenderness was present in many cases and, not infrequently,

left flank tenderness. The initial temperature ranged from 99° to 103° F., and the pulse from 60 to 160. The leukocyte count was over 15,000 in nine patients and above 10,000 in 19 patients. The urine analysis is of special interest, as 16 out of the 29 showed two plus and three plus albumin, a finding which is not generally emphasized and which undoubtedly reflects the severity of the disease. Gallstones were present in 14 surgical patients and there was roentgenographic evidence of their presence in two not operated upon, making a total of 55 per cent. In nine, the gallbladder was normal at operation or roentgenographically, while in four patients there was no data given. The patients ranged in age from 23 to 79 years, with an average age of 49. There were 21 women and eight men. Table I shows the incidence of the various symptoms and findings.

TABLE I

SUMMARY OF CHIEF SYMPTOMS AND FINDINGS

1.	Duration of symptoms 24 hours or less	19
2.	Nausea and vomiting	18
3.	Temperature 101° F., or over	18
4.	Urine albumin two plus, or over	16
5.	Gallstones present	16
6.	Leukocyte count over 15,000	9
7.	Icteric index over 15	7

Diagnosis.—The diagnosis of pancreatitis has been made in the acute surgical patients by the finding of wine-colored fluid or fat necrosis in the presence of a thickened pancreas. In the patients not subjected to operation the diagnosis has been reached by the clinical manifestations and the finding of a urinary diastase of 300 or more Foged units. The test has been accurate in our experience, and in the cases where early operation has been performed we have had no patients with a positive test in whom there has not been evidence of pancreatitis. In the patients operated upon one or two weeks after the onset, the process had regressed so that gross pathologic evidence of pancreatitis was not present. In the group of patients undergoing delayed operations, some as long as 16 days from the acute onset, the diagnosis has been made by the same criteria used in the nonoperative cases. The three delayed cases, operated upon from four to eight days from the onset of their illness, showed wine-colored fluid or fat necrosis, while the seven cases operated upon from nine to 16 days after the onset had neither fluid nor fat necrosis.

Cases of elevated diastase have been reported in patients with peptic ulcers which penetrate into the pancreas[8] and in mumps[11] pancreatitis. No cases of this type were observed in this series.

Treatment.—The method of treatment divided itself into three distinct groups of cases: 1. Those having immediate operation. 2. Those having a delayed operation. 3. Those having no operation. Nine patients were operated upon as an emergency procedure within the first 72 hours of the disease, ten were operated upon later, from the fourth to 16th day of their illness, and ten had no operation. There were four deaths in the entire group,

giving a gross mortality of 14 per cent. Three of these deaths occurred in the patients who were subjected to immediate operation, giving a mortality of 33 per cent for that group. The fourth death was in the nonoperative group, and occurred in a diabetic patient who died in uncontrollable diabetic coma, and at autopsy was found, to everyone's complete surprise, to have a fulminating pancreatitis. In the nine cases of immediate operation the correct diagnosis was not made before operation, and the following preoperative diagnoses were given: Acute cholecystitis, 4; perforated peptic ulcer, 2; chronic cholecystitis, 1; twisted ovarian cyst, 1; and acute appendicitis, 1. In the patients operated upon later, the preoperative diagnosis of acute pancreatitis was made once, and substantiated by finding fat necrosis, in the remaining cases the diagnoses were either acute or chronic cholecystitis, which were correct, as in most instances the pancreatitis had subsided at the time of operation.

The severity of the disease is a most important factor in the outcome of the patient and, in order to evaluate our operative and nonoperative results more accurately, an attempt was made to group the cases into the classifications "severe" or "mild." Patients who had three out of four of the following findings were considered severe: A temperature over 101°F., a pulse about 100, a leukocyte count over 10,000, and a urine showing two plus albumin or greater. Those not having three of the above findings were classed as mild. In the immediate operative group, five were severe; in the delayed group, four were severe; and in the nonoperative group, four were severe; so that, in all, 41 per cent of the cases were severe. Tables II, III, and IV show the classification data, the diastase level, and the type of operation performed.

Complications.—There were two cases of pancreatic cysts in this group. One was in a nonoperative patient, and reached the size of a small grapefruit in the left upper abdomen, and then spontaneously subsided. The other patient returned to be operated upon five months after her initial attack, the cyst was marsupialized, and she made a satisfactory recovery. There was one case of residual intra-abdominal sepsis which finally drained through in the original drainage wound. One patient had two attacks of pancreatitis and finally, on a third admission, cholecystotomy was performed during a quiescent period of his disease.

Discussion.—There is no doubt that progress has been made in the diagnosis and in the treatment of acute pancreatitis. It is becoming more and more evident that hasty operative intervention in the very acute phase of the disease is a hazardous procedure. The evidence at hand of a 33 per cent mortality in the immediate operative group, and a 5 per cent mortality in combined nonoperative and delayed operative groups should make the surgeon deliberate carefully before contemplating celiotomy in acute pancreatitis. The diastase test is of invaluable assistance in arriving at a diagnosis. If operation is necessary it is best undertaken during the less severe phase of the disease, for the drainage of residual abscess

TABLE II

OPERATIONS WITHIN 72 HOURS FROM ONSET OF SYMPTOMS
Recovered 6—Died 3

History Number	Age	Temperature 101° F. Plus	Pulse 100 Plus	W. B. C. 10,000	Urine Albumin 2—Plus	Urine Diastase Units	Duration before Operation	Operation	Result
				SEVERE					
412-968	72	101	100	Not made	2 plus	380	2 hours	Drain to pancreas	
389-017	48	103	100	27,000	1 plus	600	12 hours	Cholecystostomy	
357-761	57	101	120	34,000	3 plus	Not made	24 hours	Cholecystostomy Drain to pancreas	Died
386-499	38	101	100	15,900	2 plus	570	72 hours	Abdomen drained	Died
365-156	51	103	160	15,800	2 plus	1020	24 hours	Drain to pancreas	Died
				MILD					
387-952	44	98.6	80	31,000	2 plus	240*	6 hours	Cholecystectomy	
371-566	41	101	90	19,950	0	540	48 hours	Cholecystectomy	
375-098	52	102	80	19,000	1 plus	120*	24 hours	Appendicectomy	
374-403	53	98	100	Not made	2 plus	Not made	6 hours	Cholecystectomy	

*Test made six days postoperative.

Note: Fat necrosis or wine-colored ascitic fluid was found at operation in all of the above cases.

TABLE III

OPERATIONS AFTER 72 HOURS FROM ONSET OF SYMPTOMS
Recovered 10—Died 0

History Number	Age	Temperature 101° F. Plus	Pulse 100 Plus	W. B. C. 10,000	Urine Albumin 2—Plus	Urine Diastase Units	Duration before Operation	Operation
				SEVERE				
416-448.....	61	101	100	8,500	2 plus	540	8 days	Cholecystectomy
393-054.....	27	102	120	10,500	3 plus	2,400	4 days	Cholecystectomy
387-537.....	23	101	100	20,200	2 plus	600	8 days	Cholecystectomy
364-896.....	47	102	100	14,200	2 plus	1,020	9 days	Cholecystectomy
				MILD				
372-379.....	39	103	110	5,400	1 plus	960	16 days	Cholecystectomy. Choledochostomy
360-675.....	57	99	70	11,900	1 plus	120*	4 days	Drain to pancreas
414-522.....	79	100	90	13,500	2 plus	300	15 days	Cholecystostomy
418-868.....	23	100	100	11,500	1 plus	2,400	16 days	Cholecystectomy
387-386.....	60	100	80	9,300	1 plus	1,275	4 days	Cholecystostomy
383-329.....	44	101	90	5,600	1 plus	1,020	11 days	Cholecystectomy. Choledochostomy

*Test made ten days postoperative. Fat necrosis found at operation.

TABLE IV

NONOPERATIVE CASES
Recovered 9—Died 1

History Number	Age	Temperature 101° F. Plus	Pulse 100 Plus	W. B. C. 10,000	Urine Albumin 2—Plus	Urine Diastase Units	Duration before Entry	
				SEVERE				
381-998.....	59	103	100	19,000	1 plus	480	10 hours	
423-159.....	57	102	100	not made	2 plus	540	11 days postop.‡	
406-412 399-186 *.....	58	101	100	11,300	0	2640	5 hours	
359-529†.....	54	100.5	110	not made	2 plus	not made	not given	Died
				MILD				
412-433.....	60	100	70	12,800	2 plus	1020	5 days	
397-891.....	62	100	120	8,400	3 plus	2100	24 hours	
415-089.....	36	101	120	9,300	1 plus	2250	48 hours	
363-601.....	23	102	86	10,300	0	630	24 hours	
371-825.....	40	99	90	10,850	2 plus	510	7 days	
399-186.....	58	103	100	8,000	1 plus	1800	4 hours	

*Patient had two admissions, both for pancreatitis.

†Due to severe diabetes with coma, this patient failed to show the normal systemic reactions to her disease. Diagnosis made at autopsy.

‡Patient developed an acute pancreatitis during convalescence from cholecystectomy.

or cyst. The frequent association of pancreatitis with cholecystitis often leads to later operations upon the gallbladder and bile ducts.

SUMMARY AND CONCLUSION

1. Twenty-nine cases of acute pancreatitis seen at the Hartford Hospital, from September 1, 1938 to December 31, 1941, have been presented.

2. In the cases subjected to immediate operation the mortality was 33 per cent.

3. In the patients treated with delayed operation and without operation the mortality was five per cent.

4. Conservative management in cases of acute pancreatitis is recommended.

REFERENCES

[1] Fitz, R. H.: Acute Pancreatitis. Boston Med. and Surg. Jour., **120**, 205-229, 1889.

[2] Opie, E. L.: Disease of the Pancreas. 1903—J. B. Lippincott Co., Philadelphia.

[3] Wohlgemuth, J.: Contributions on Functional Diagnosis of the Pancreas. Berl. klin. Wchnschr., **47**, 92–95, January 17, 1910.

[4] Mikkelsen, Otto: Acute Pancreatitis. Acta chir. Scandin., **75**, 373-415, 1934.

[5] Henderson, F. F., and King, E. S. A.: Acute Pancreatitis. Arch. Surg., **30**, 1049-1057, 1935.

[6] Lium, R.: Diagnosis and Conservative Treatment of Acute Pancreatitis. N. E. Jour. of Med., **219**, 881-885, December 1, 1938.

[7] Fallis, L. S., and Plain, G.: Acute Pancreatitis. Surgery, **5**, 358-373, March, 1939.

[8] Lewison, E. F.: Acute Pancreatitis. Arch. Surg., **41**, 1008-1037, October, 1940.

[9] Lewison, E. F.: The Clinical Value of the Serum Amylase. Surg., Gynec., and Obst., **72**, 202-212, February, 1941.

[10] Abell, I.: Acute Pancreatitis. Surg., Gynec. & Obstet., **66**, 348-353, February 15, 1938.

[11] Elman, R.: Surgical Aspects of Acute Pancreatitis. J.A.M.A., **118**, 1265-1270, April 11, 1942.

[12] Foged, J.: The Diagnostic Value of Urine Diastase. Am. Jour. Surg., **27**, 439-446, March, 1935.

PULMONARY ABSCESS—A SURGICAL PROBLEM*

CLASSIFICATION OF CASES AND DISCUSSION OF SURGICAL TREATMENT

RICHMOND L. MOORE, M.D.
NEW YORK, N. Y.

FROM THE DEPARTMENT OF SURGERY, COLUMBIA UNIVERSITY, AND THE SURGICAL SERVICE OF THE PRESBYTERIAN HOSPITAL, NEW YORK, N. Y.

PULMONARY ABSCESSES are commonly classified as acute or chronic, the distinction between the two being made solely on the basis of time. This practice has led to much confusion because the terms, acute and chronic, are vague in themselves and because the interval of time chosen to separate the two varieties of abscess represented by these terms is not always the same. Confusion is seen especially in the interpretation of statistics which are supposed to show the difference in the results of treatment between the so-called acute and chronic types. My belief that a pathologic classification is of far greater value than a clinical one, and that I could show this by analyzing the experience gained in treating 78 patients, has prompted me to prepare this report. Furthermore, it has been my feeling for some time that exact pathologic differentiation of pulmonary abscess deserved greater emphasis because of the satisfactory results which are now being obtained by lobectomy in an increasing number of selected cases. In this report, my cases have been divided into two main groups which are designated *uncomplicated* and *complicated*. By definition, a case was considered uncomplicated when there was only one abscess and this was unassociated with empyema, notable fibrosis, bronchiectasis or other serious complications. When there were multiple abscesses, empyema or pyopneumothorax, notable fibrosis, bronchiectasis, or other more rare complications such as suppurative pericarditis and supperative mediastinitis, the cases were classified as complicated.

By *multiple abscesses* is meant two or more separate and distinct abscesses, or multiple cavities, in the same part of a lung which are connected by such small channels that something more than a simple unroofing operation was required in order to open them. The term does not necessarily imply secondary abscesses because occasionally two or more abscesses develop simultaneously in different parts of the same lung, or in both lungs.

The *empyemata*, all proved by operation, were recognized as complications of coexisting abscesses only when the clinical or roentgenologic evidence of abscess was unmistakable, or when an abscess was demonstrated at operation or subsequently during the postoperative course.

Notable fibrosis was assumed to be present when operation revealed an epithelialized cavity with rigid, irregular wall and multiple fistulae (so-called *"gitter lunge"*) or when a cavity with rigid wall was found in a case with or without roentgenologic evidence of fibrosis, such as shrinkage or atelectasis of the involved lung, with displacement of the mediastinum and fixation of the chest wall.

Bronchiectasis was considered to be present only when it was demonstrated by bronchography or by direct examination of material removed at operation or necropsy. For the

* Read before the New York Surgical Society, April 22, 1942.

sake of simplicity, the patients with more than one complication were classified under only one heading, preference being given to the complications present in the following order: Bronchiectasis; empyema; multiple cavities; and notable fibrosis.

The division of abscesses into uncomplicated and complicated groups on the basis of pathology, is contrary to the common practice of classifying them as "acute" and "chronic," on the basis of duration of symptoms. According to this, the usual classification, an acute abscess is one of short duration with a wall composed of soft, necrotic tissue; whereas a chronic abscess is one of longer duration which is surrounded by a dense zone of fibrous tissue, with, in most instances, some degree of shrinkage and bronchiectasis in the involved part of the lung. The interval of time which is supposed to differentiate the two varieties is arbitrary and varies with different writers. It is usually given as six to 12 weeks. This clinical differentiation between the two varieties of abscess, on the basis of a time factor, puts one in the absurd position of having to say that an abscess changes from one type to the other overnight. It also necessitates the selection of a certain day for the beginning of every abscess, whereas in some instances the history of onset is so vague that it is impossible to determine with any accuracy when the abscess originated. Furthermore, even when the approximate time of onset is known, a time-interval alone, whether it be six weeks, eight weeks, or 12 weeks, is not a reliable criterion. There will always be some abscesses which drain poorly and become associated with fibrosis and bronchiectasis before the elapse of this time and others that drain well and remain uncomplicated for a longer period. Furthermore, there are probably other factors which determine the development of these complications, such as the type and virulence of the infecting agent and the resistance of the patient.

These are my reasons for disliking a clinical classification based on time. It is inaccurate. On the contrary, a classification based on pathology directs attention at once to the complications which are so frequently associated with pulmonary abscess and which have such an important influence on the outcome.

BACTERIOLOGY

In spite of extensive investigation the relative importance of different bacteria in the development of pulmonary abscess is still unknown. Clinically, however, two types of lung abscess *can* be distinguished. One of these, which is by far the more common, is associated with a very foul odor. The other is not. The odor is striking and can usually be recognized at the bedside, when the abscess discharges its contents through a bronchus, or in the operating room, when the abscess is opened surgically. This foul type of abscess is now generally believed to represent an anaerobic infection of the lungs, caused by organisms which are commonly found in dirty mouths. The anaerobes which are thought to be of greatest importance include the Spirochaeta and fusiform bacilli of Vincent, the Bacterium melaninogenicum, Vibriones and streptococci.

Some authors, notably Wessler[1] and Neuhof,[2] believe that the odor is diagnostic and always represents an anaerobic infection. They also believe that the lesion produced represents a peculiar type of pathology which is characterized by mass tissue necrosis, or gangrene. This may be localized to a small area or spread to involve an entire lobe. Such lesions are called "gangrene of the lung" by Wessler and, when localized, "putrid lung abscess" by Neuhof. Both of these authors distinguish them from the more rare "second type" of lung abscess which is not fetid and which is found in association with a wide variety of aerobic pyogenic bacteria.

In this report the fetid and nonfetid varieties of abscess are discussed separately because, in spite of the uncertainty as to the causal relationship of various bacteria, I also believe that an abscess which is fetid in its early stages represents an anaerobic infection of the lungs and presents a different problem in therapy than is presented by a nonfetid abscess. It was considered futile to attempt a bacteriologic classification when the exact part played by different bacteria is still uncertain. Furthermore, this would have been impossible, because complete bacteriologic studies, including darkfield examinations and anaerobic cultures, were not made in all of the cases, and most of the cases were of many months' standing when they first came under my observation. Obviously, the bacteria recovered from an abscess of many months' duration are not necessarily the same as those which initiated the process.

Even though the exact importance of anaerobic bacteria in the development of an abscess has not been established to the satisfaction of all, the results of the bacteriologic studies made on the patients with fetid abscess, which are included in this report, would certainly seem to be of some significance. In spite of the fact that complete studies were not made in all instances, either anaerobic streptococci, Bacterium melaninogenicum, Vibriones, Spirochaeta or fusiform bacilli, singly or in varying combinations, were recovered from the sputum, abscess contents, or empyema fluid in 42 of the 70 fetid cases in the series. The organisms recovered in the nonfetid cases included the Friedländer bacillus in one case, Aspergillus fumigatus in one, pneumococcus Type-III in one, hemolytic *Staphylococcus aureus* in one, and hemolytic streptococcus in two. Bacteriologic studies in the other two nonfetid cases were unsatisfactory.

PATHOGENESIS

There are several etiologic factors which are important both from the diagnostic and therapeutic standpoints. For instance, the history of onset in the majority of fetid abscesses suggests that they are due to the aspiration of infected material from the mouth. A great many are postoperative, and most of these occur after operations upon the upper respiratory tract, such as tonsillectomies, laryngectomies, tooth extractions and drainage of abscesses. Some of the cases that occur following other types of operations are clearly the result of secondarily infected infarcts. Not a few fetid abscesses occur

after periods of unconsciousness due to alcohol, drowning accidents, insulin shock, trauma, or epileptic seizures. In this group there is sometimes a definite history of the aspiration of vomitus, water, or some other foreign material. In contrast, the history of onset in most of the nonfetid abscesses suggests an embolic or postpneumonic origin. Both types occur, however, without a history of antecedent trauma, operative procedure, or pneumonia. Thus, it should be emphasized that, even if an etiologic factor predisposing to aspiration is absent, fetid abscesses are probably aspiration abscesses and represent infection either primary or secondary, caused by the group of anaerobic organisms mentioned above.

PATHOLOGY AND CLASSIFICATION

In its early stages a pulmonary abscess may be looked upon as a localized area of necrosis and suppuration which is unassociated with any significant amount of fibrosis and bronchiectasis. Of all the factors concerned in the healing of such a process free drainage is by far the most important. This may occur spontaneously through a bronchus or may be established by operation. In either event the result may be equally good. If free drainage is not obtained early, however, certain complications and permanent changes in the adjacent lung are likely to occur, both secondary to the persisting infection. The usual complications are a spreading pneumonitis, bronchopneumonia, secondary abscesses and empyema. The permanent changes in the adjacent lung are fibrosis and bronchiectasis. The presence of fibrosis and bronchiectasis is generally understood as establishing the difference between an acute and chronic process.

When a lung abscess is complicated by empyema or pyopneumothorax it usually means that the abscess has perforated into the pleural cavity and that a bronchopleural fistula exists (so-called pleuropulmonary abscess). This is not an uncommon complication of abscess and, in many instances, the fistula can be demonstrated, either at operation, or subsequently during the postoperative course. Empyema may also follow a diagnostic aspiration of a lung abscess from contamination of the pleural cavity by the needle. Since this does not seem to be an infrequent occurrence it is felt that a deliberate lung puncture should never be made in a case of suspected abscess, and that diagnostic punctures, in general, should be avoided whenever possible, for fear of accidental lung injury and pleural infection. The danger of infecting the pleural cavity by thoracentesis seems to be greater in the anaerobic infections. When empyema is a complication of a fetid abscess the exudate is foul and usually much thinner than the exudate which is seen in the more familiar pneumococcal and streptococcal varieties of empyema. Cultures reveal the same anaerobic organisms which are found in association with fetid abscesses. Empyemata similar to these, and associated with the same organisms, are occasionally seen when there is no evidence whatsoever of either a preceding or coexisting abscess. The origin of the empyema in cases of this kind is obscure. There is reason to believe that some foul

empyemata are caused by anaerobic streptococci alone. These cases appear to be postpneumonic and the exudate is thick, not unlike that seen in the pneumococcal and hemolytic streptococcal infections. Occasionally the presence of a lung abscess is never suspected until foul pus is aspirated from the pleural cavity. When empyema occurs as a complication of lung abscess it may be a small, well-encapsulated collection of pus, or a large, poorly circumscribed collection occupying the greater part of the pleural space. The smaller encapsulated forms are found, not infrequently, in one of the interlobar spaces, and I have the impression that they are seen especially in association with upper lobe abscesses. They suggest slow leakage from the underlying abscess, allowing time for protective adhesions to form. The massive types suggest sudden rupture into a virgin pleural cavity.

TREATMENT

Uncomplicated Abscess—Fetid Type.—In common with Wessler, Neuhof, and many others, I feel that a fetid abscess is essentially a surgical disease. This is not meant to imply that all cases require operation. On the contrary, I believe that all cases should have a trial of conservative therapy, because some will drain freely through the bronchial tree and heal satisfactorily without surgical interference. The majority, however, do not, and for this reason, during the period of conservative treatment, the patients should be under the closest supervision of both the internist and the consulting surgeon. When evidence of free drainage and progressive healing becomes questionable, surgical drainage should be instituted at once, unless there is some very good reason for not doing so. *Early free drainage is essential to avoid the development of complications and the higher operative mortality and less satisfactory late results in the complicated cases.* Unfortunately, the medical profession, at large, does not seem to appreciate this, for in most clinics it is still true that the majority of cases are not referred for surgical treatment until after complications have developed. In the series of abscesses herewith reported, 54 out of 78, or 69 per cent, fell into this group.

Precise localization of the cavity is by far the most important technical factor in determining the success of the operation. When the abscess is situated in the peripheral part of a lobe, as most of them are, adhesions form early between the overlying visceral and parietal layers of pleura and make drainage of the abscess possible in a one-stage procedure without opening and infecting the pleural cavity. In some instances, however, adhesions are not found, either because the surgical approach is not correct or because the cavity is central and unassociated with pleural adhesions. Under these conditions the operation must be undertaken in two stages. At the first stage the wound is packed with iodoform gauze in order to produce the desired adhesions artificially. It is important to place the iodoform gauze in close contact with the parietal pleura. The abscess is opened six to eight days later. In the interval, roentgenologic studies to localize the cavity should be repeated. The number of operations that have to be divided into stages should

decrease as a surgeon becomes more proficient in localization. In general, upper and middle lobe abscesses are more directly drained through an anterior or axillary approach and lower lobe abscesses through a posterior approach. Local anesthesia is the anesthesia of choice in all cases.

Uncomplicated Abscess—Nonfetid Type.—Early drainage is also of importance in this group although perhaps less so than in the fetid abscesses. This is because the associated aerobic bacteria are less destructive than the anaerobic bacteria found in the fetid cases and are less prone to cause secondary abscesses and empyema. On the other hand, early drainage to avoid secondary fibrosis appears to be equally as important. Nonfetid abscesses are relatively rare in comparison with fetid abscesses and are often unsuitable for operation because of their extent and the presence of other septic foci. The operative technic in these cases is the same as that in the fetid cases.

Complicated Abscess—Fetid and Nonfetid Types with Multiple Cavities.—Multiple cavities may require drainage at two separate sites. When they involve the same bronchopulmonary segment, however, usually a single approach will suffice. If the "several cavities" prove to be one large multilocular cavity, and the smaller chambers drain freely into the main space, a simple unroofing operation of the main cavity may provide the necessary drainage. Otherwise, it may be necessary to unroof each cavity separately.

Complicated Abscess—Fetid and Nonfetid Types with Empyema.—When an abscess is complicated by empyema, the empyema should be drained first. In most cases drainage of the empyema will suffice because the abscess drains freely into the empyema cavity at the site of rupture. In the fetid cases it is important to operate immediately because of the marked sepsis usually present in these infections and in order to avoid the development of a cellulitis of the chest wall from bacteria implanted in the needle tract at the time of thoracentesis. When the empyema is small and well-encapsulated, drainage should be instituted through a large opening into the dependent part of the cavity. When, on the other hand, the empyema cavity is large and poorly circumscribed, an open thoracotomy may be hazardous. In cases of this kind the opening into the pleural cavity should be large, but measures should be taken to prevent a "sucking" wound until the desired adhesions have formed. If there is a fistula, continuous suction might help in preventing the aspiration of exudate from the empyema cavity, with spread of infection to other bronchi. Swabbing or irrigating the cavity with an aqueous solution of gentian violet seems to help in some cases. Whether this is due to the prevention of secondary infection or to a specific effect of the gentian violet on the organisms which were primarily responsible for the abscess is not clear. When there is no fistula other antiseptics such as Dakin's solution or zinc peroxide may be used. When improvement does not follow drainage of the empyema, inadequate drainage of the underlying abscess should be suspected. Other likely causes are pulmonary gangrene, suppurative mediastinitis and brain abscess. Empyema is rarely seen in association with a proved nonfetid abscess. It did not occur once in the eight cases of nonfetid abscess

included in this report, whereas the instance of empyema in the fetid cases was 23 per cent.

Complicated Abscess—Fetid and Nonfetid Types with Notable Fibrosis and Bronchiectasis.—The treatment of lung abscess with secondary fibrosis and bronchiectasis is the same, regardless of whether the preceding abscess was fetid or nonfetid. Conservative measures will not produce healing in cases of this kind. Surgery offers the only hope of cure and, here again, the first consideration should be efficient drainage of the involved part of the lung. The rationale for this is twofold. In the first place, occasionally such a case will heal after free drainage is established; and in the second place, those that do not heal are improved by drainage and hence become better risks for what further procedures are indicated. If there is a minimal amount of bronchiectasis, satisfactory drainage can often be obtained by simply unroofing the cavity. On the other hand, when there is advanced bronchiectasis efficient drainage can only be established by destroying some of the involved part of the lung. The cautery is the best means for accomplishing this.[3] Both the electric and actual cauteries can be used. Experience seems to have shown, however, that the latter is less likely to be associated with hemorrhage and air embolism and is, therefore, preferable. The use of the cautery to secure drainage is best suited to peripheral lesions that involve only one lobe of a lung. When a large part or all of a lobe is involved the amount of lung tissue which must be destroyed in order to obtain satisfactory drainage is so large that spontaneous healing seldom follows. The result may be a single fistula, but in most instances it will be a residual cavity in the lung itself with multiple bronchial communications. Fibrous partitions of varying height divide the walls of such a cavity into recesses which, in time, may become completely covered by epithelium. Patients with pathology such as this are often in the best of health except for their fistulae. Another surgical procedure is necessary, however, in order to produce healing. This should be a plastic operation, with excision of the mucous membrane lining the cavity and closure of the fistulae if the central part of the lobe is normal.[4] Lobectomy is the better procedure when the remnant of cauterized lobe is fibrotic and bronchiectatic.

When fibrosis and bronchiectasis involves more than one lobe, cautery pneumonectomy is less practical. In cases of this kind immediate removal of all of the diseased part of the lung is the only rational procedure. This may require a bilobar lobectomy, or total pneumonectomy. It should be realized, however, that the mortality of such procedures is high, especially so in the presence of residual pus in patients with fever, leukocytosis, debility, cyanosis and copious amounts of sputum. The use of suction to remove exudate from the trachea and bronchi before, during, and immediately after operation upon patients of this kind is of great importance.

CLINICAL MATERIAL

This report is based on the records of 78 consecutive patients who were treated surgically at the Presbyterian Hospital during the past 11 years.

Fifty-one were males; 27 were females. Of the 78 patients, only 24 had uncomplicated abscesses at the time surgery was instituted, leaving 54 patients who had already developed complications. In the uncomplicated group, 23 of the cases were fetid; and 1 was nonfetid. In the complicated group, 47 of the cases were fetid and 7 were nonfetid. Of the 47 patients with fetid abscess and complications, 14 had multiple cavities; 16 had empyema; 2 notable fibrosis of the lung; 13 bronchiectasis; 1 a mediastinal abscess; and 1 an abscess of the kidney. All of the patients with nonfetid abscess and complications had extensive fibrosis of the lung (Table I).

TABLE I

CLINICAL MATERIAL

Seventy-eight Patients with Pulmonary Abscess

I. Uncomplicated		24
A. Fetid	23	
B. Nonfetid	1	
II. Complicated		54
A. Fetid	47	
Multiple cavities	14	
Empyema	16	
Notable fibrosis	2	
Bronchiectasis	13	
Mediastinal abscess	1	
Abscess of the kidney	1	
B. Nonfetid	7	
Multiple cavities	0	
Empyema	0	
Notable fibrosis	7	
Bronchiectasis	0	

When the patients were grouped according to duration of symptoms it was found that only 31 had had symptoms for less than 2 months at the time of operation. Of these, 18, or 58 per cent, had already developed complications. The 18 included 4 patients with multiple cavities; 13 with empyema; and 1 with bronchiectasis. When the interval of time was extended to 3 months the percentage of complicated cases was the same. There were 40 patients in this group, 6 of whom had multiple cavities; 14 empyema; 1 notable fibrosis; 1 bronchiectasis; and 1 a mediastinal abscess. After this, the percentage of patients with complications increased with the duration of symptoms but the type of complication changed. When the duration of symptoms exceeded 3 months (38 patients), 31, or 82 per cent, had complications. In this group, however, there were only 2 cases with empyema. Eight of the others had multiple cavities; 8 notable fibrosis; 12 bronchiectasis; and 1 a sinus extending through the diaphragm into an abscess of the kidney (Table II).

RESULTS

Uncomplicated Abscess—Fetid Type.—Of the 23 patients with uncomplicated abscess of the fetid type 21 recovered. All of these are still alive and have been followed for periods of time varying from a few months to 11 years. Nineteen have healed wounds and are free of symptoms. Two are healed, with a residual cough. The mortality in this group (2 deaths) was

TABLE II

CLINICAL MATERIAL

Seventy-eight Patients with Pulmonary Abscess

1. With symptoms not exceeding 2 months			31
Uncomplicated		13	
Complicated		18 (58%)	
Multiple cavities	4		
Empyema	13		
Bronchiectasis	1		
2. With symptoms not exceeding 3 months			40
Uncomplicated		17	
Complicated		23 (58%)	
Multiple cavities	6		
Empyema	14		
Notable fibrosis	1		
Bronchiectasis	1		
Mediastinal abscess	1		
3. With symptoms for more than 3 months			38
Uncomplicated		7	
Complicated		31 (82%)	
Multiple cavities	8		
Empyema	2		
Notable fibrosis	8		
Bronchiectasis	12		
Abscess of kidney	1		

9 per cent. One patient died on the fifth day from a pneumonia, which appeared to be due to the aspiration of exudate which spilled into the air passages either during or soon after the operative procedure. The other death was apparently caused by an acute agranulocytosis. This patient, a male diabetic, age 58, had a large abscess of the middle lobe. He was treated vigorously with sulfapyridine before transfer for surgery, and at the time of operation had a leukopenia. At operation, a large piece of sequestrated lung tissue was removed from the abscess cavity. The agranulocytosis developed immediately after operation. The patient ran a high temperature, showed no tendency to improve, and died on the fourth postoperative day. The results are summarized in Table III, and the surgical procedures are given in Table IV.

TABLE III

UNCOMPLICATED FETID ABSCESS

Mortality and Late Results in 23 Consecutive Patients

Recovered			21
Healed, and symptom-free		19	
Followed 0–2 years	9		
Followed 2–4 years	4		
Followed 4–6 years	5		
Followed 10 years 9 months	1		
Healed, with slight symptoms remaining		2	
Followed 3 years 6 months	1		
Followed 9 years 1 month	1		

Mortality—9%

Uncomplicated Abscess—Nonfetid Type.—Bacteriologic study of the exudate taken from the abscess cavity at the time of operation, in the one patient who had an uncomplicated abscess of the nonfetid type, revealed a pure

TABLE IV

UNCOMPLICATED FETID ABSCESS

Surgical Procedures in 23 Consecutive Patients

Rib resection and drainage, in 1 stage	14
Rib resection and drainage, in 1 stage, followed by closure of fistula	1
Rib resection and drainage, in 2 or more stages	8

culture of Friedländer bacillus. This abscess was drained in two stages. The patient made an uneventful recovery, and was completely relieved of symptoms. Follow-up observations continued until his death from carcinoma of the esophagus, six and one-half years later.

Complicated Abscess—Fetid Type, with Multiple Cavities.—In this group there were 14 patients. Of these, 9 recovered, and have been followed for periods of time varying between 9 months and 7 years. Five of the 9 have healed wounds, and are symptom-free; one has a residual cavity, with fistulae; one is healed, with slight symptoms; one still has a bronchocutaneous fistula after two and one-half years; and the other, also with a bronchocutaneous fistula, died of a streptococcal wound infection and septicemia following appendicectomy, 22 months after the surgical attack on the abscess was completed. The mortality in this group (5 deaths) was 36 per cent. One patient died during operation, death apparently being related to the administration of the anesthetic. Another died of a massive hemorrhage from the lung, two and one-half years after drainage of a large middle lobe abscess. Another, a male diabetic, age 59, died of unknown cause a few weeks after discharge from the hospital. The other two died of pneumonia, 1 eight days after operation, and the other two years after operation. The results are summarized in Table V, and the surgical procedures in Table VI.

TABLE V

COMPLICATED ABSCESS—FETID TYPE, WITH MULTIPLE CAVITIES

Mortality and Late Results in 14 Patients

Recovered		9
Healed, and symptom-free		5
Followed 9 months	1	
Followed 1–2 years	2	
Followed 2 years 4 months	1	
Followed 7 years 4 months	1	
Residual cavity, with fistulae		1
Followed 1 year 6 months		
Healed, with slight symptoms remaining		1
Followed 1 year 9 months		
Bronchocutaneous fistula		2
Followed 1 year 10 months	1	
Followed 2 years 6 months	1	

Mortality—36%

TABLE VI

COMPLICATED ABSCESS—FETID TYPE, WITH MULTIPLE CAVITIES

Surgical Procedures in 14 Patients

Rib resection and drainage, in 1 or more stages	6
Rib resection and drainage, followed by lobectomy	1
Cautery pneumonectomy, in stages	4
Cautery pneumonectomy, followed by closure of fistula	1
Cautery pneumonectomy, followed by unsuccessful closure of fistula	1
Pneumonectomy attempted but not completed	1

Complicated Abscess—Fetid Type, with Empyema.—Seventeen of the patients with fetid abscesses had an associated empyema or pyopneumothorax. One of these, with symptoms of more than a year's duration, had, in addition, extensive fibrosis and bronchiectasis of the involved lung, and is included in the group with these complications. Of the remaining 16 patients, 11 recovered, and all of these have been followed to the present time. Ten of the 11 have healed wounds, and are symptom-free; and 1 has a bronchocutaneous fistula. The mortality (5 deaths) was 31 per cent. Two of the patients who died developed brain abscesses. Another developed an extensive necrotizing cellulitis of the chest wall and lumbar region. A fourth developed large areas of gangrene in both lower lobes. The fifth death, which came suddenly on the fourth day after operation, was apparently caused either by a massive cardiac infarction or pulmonary embolism. The duration of symptoms previous to operation was less than six weeks in ten of the patients. The results are shown in Table VII, and the surgical procedures in Table VIII.

TABLE VII

COMPLICATED ABSCESS—FETID TYPE WITH EMPYEMA

Mortality and Late Results in 16 Consecutive Patients

Recovered		11
Healed, and symptom-free	10	
Followed 0–1 year	2	
Followed 1–2 years	4	
Followed 2–3 years	2	
Followed 3 years 6 months	1	
Followed 4 years 4 months	1	
Bronchocutaneous fistula		1
Followed 7 months		

Mortality—31%

TABLE VIII

COMPLICATED ABSCESS—FETID TYPE WITH EMPYEMA

Surgical Procedures in 16 Consecutive Patients

Rib resection, with drainage of empyema only	14
Rib resection, with drainage of empyema, followed by drainage of abscess	2

Complicated Abscess—Fetid Type, with Fibrosis of the Lung and Bronchiectasis.—In this group there were 15 patients; 13 with proved bronchiectasis; and two with fibrotic lobes, without demonstrable bronchiectasis cavities. Only 1 of the 15 patients had had symptoms for less than three months. In 8, the duration of symptoms exceeded one year, and in the other 6, symptoms had been present for periods of time varying between four and ten months. In reporting results, the two patients with pulmonary fibrosis, but without demonstrable bronchiectasis, have been grouped with the 13 with bronchiectasis because of my belief that these two conditions rarely, if ever, occur separately in fetid cases of pulmonary suppuration. Ten of the patients recovered, and all of these have been followed for varying periods of time. Only four have healed wounds and are symptom-free. Two are healed, with slight symptoms. Four have bronchocutaneous fistulae. The mortality

(5 deaths) was 33 per cent. Three of the deaths were apparently caused by a postoperative pneumonia, resulting from the aspiration of exudate which spilled into the air passages either during or soon after the operative procedure. One death was due to a severe hemorrhage, which occurred during operation, when the stump of an amputated lobe slipped out of the lobectomy cord. The other patient died on the fifth postoperative day of extensive infection including secondary abscesses in the opposite lung, a severe purulent bronchitis and empyema. Results are shown in Table IX, and operative procedures in Table X.

TABLE IX

COMPLICATED ABSCESS—FETID TYPE, WITH FIBROSIS AND BRONCHIECTASIS

Mortality and Late Results in 15 Patients

Recovered		10
Healed, and symptom-free		4
Followed 2 years 9 months	1	
Followed 4 years 7 months	1	
Followed 6 years 6 months	1	
Followed 8 years 5 months	1	
Healed, with slight symptoms remaining		2
Followed 5 years 8 months	1	
Followed 5 years 9 months	1	
Bronchocutaneous fistula		4
Followed 4 months	2	
Followed 1 year 8 months	1	
Followed 5 years 9 months	1	

Mortality—33%

TABLE X

COMPLICATED ABSCESS—FETID TYPE, WITH FIBROSIS AND BRONCHIECTASIS

Surgical Procedures in 15 Patients

Rib resection and drainage, in 1 or more stages	3
Rib resection and drainage, followed by closure of fistula	1
Cautery pneumonectomy, in stages	4
Cautery pneumonectomy, followed by lobectomy	6
Primary lobectomy	1

Complicated Abscess—Nonfetid Type, with Notable Fibrosis of the Lung.—There were seven patients in this group, and all had had symptoms for months or years before the surgical attack was begun. Six recovered, and have been followed for varying periods of time. Four of these have healed wounds, and are symptom-free. Two have open wounds, with residual cavities in the lung and multiple fistulae. The one death occurred on the fifth day postoperative from coronary thrombosis. Mortality and late results are summarized in Table XI, and surgical procedures are given in Table XII.

TABLE XI

COMPLICATED ABSCESS—NONFETID TYPE, WITH FIBROSIS

Mortality and Late Results in 7 Patients

Recovered		6
Healed, and symptom-free		4
Followed 5 months	1	
Followed 5–6 years	3	
Open wounds, with residual cavity and fistulae		2
Followed 2 years 2 months	1	
Followed 4 years	1	

Mortality (1 death)—14%

TABLE XII

COMPLICATED ABSCESS—NONFETID TYPE, WITH FIBROSIS AND BRONCHIECTASIS

Surgical Procedures in 7 Patients

Rib resection and drainage, in 1 stage	4
Rib resection and drainage, followed by closure of fistula	1
Rib resection and drainage, followed by lobectomy	1
Primary lobectomy	1

All Cases—Uncomplicated and Complicated.—Of the 78 patients operated upon, 43, or 55.1 per cent, are apparently cured, *i.e.*, they have healed wounds, and are entirely free of symptoms; 10, or 12.8 per cent, have bronchial fistulae; 5, or 6.4 per cent, have healed wounds, with persistent cough and sputum; and 20, or 25.7 per cent, are dead. The results were not tabulated according to procedure because of the great variation in the extent of the pathologic processes in the different patients, and the small number of patients in any particular group. None of the patients were exactly alike, and none of them with the same complications presented the same operative risk.

SUMMARY

(1) The bacteriology, pathogenesis, pathology and treatment of pulmonary abscess are discussed, and the records of 78 patients who were treated surgically by the author were reviewed. The cases were classified as *uncomplicated* or *complicated*, according to pathology, and as *fetid* or *nonfetid*, according to odor. By definition, a case was considered *uncomplicated* when there was only one abscess, and this was unassociated with empyema, notable fibrosis of the lung, bronchiectasis, or other serious complications. Cases with multiple abscess, empyema, notable fibrosis, bronchiectasis, or other rarer complications such as suppurative pericarditis and suppurative mediastinitis were classified as *complicated*. The criteria for classification are given in detail. The common practice of describing abscesses as "acute" and "chronic," according to duration, is condemned because the terms *acute* and *chronic* do not have exact meanings, and have led to much confusion.

(2) Of the 78 patients, only 24 had uncomplicated abscesses. Of the 54 with complications, 16 had empyema; 14 multiple cavities; 13 bronchiectasis; 9 notable fibrosis of the lung; 1 a mediastinal abscess; and 1 an abscess of the kidney. In the uncomplicated group, 23 of the cases were fetid; and 1 was nonfetid. In the complicated group, 47 of the cases were fetid; and 7 were nonfetid. Fifty-eight per cent of the patients who had had symptoms for less than three months, at the time of operation, were complicated; and 82 per cent of the patients who had had symptoms for more than three months, were complicated. In the fetid group, empyema and multiple abscesses were frequently seen in the early stages, whereas fibrosis and bronchiectasis, with or without secondary abscesses and empyema, were regularly seen in the cases of many months' standing. In the nonfetid group none of the cases had empyema or bronchiectasis, but all of the cases of long standing had unmistakable evidence of extensive pulmonary fibrosis.

(3) Of the 24 patients with uncomplicated abscess, only 2 died, an operative mortality of 8 per cent. In contrast, the operative mortality in the 54

patients with complications (18 deaths) was 33 per cent. In this latter group, composed largely of fetid cases, the mortality in the fetid cases with multiple cavities was 36 per cent; in the fetid cases with empyema it was 31 per cent; and in the fetid cases with fibrosis and bronchiectasis it was 33 per cent. In the nonfetid cases, all of whom had extensive pulmonary fibrosis, and none of whom had empyema or bronchiectasis, it was 14 per cent (1 death). The surgical procedures are given.

(4) All of the survivors were followed for periods of time varying from a few months to 11 years. The late results were better in the uncomplicated group and in the fetid group that was complicated by empyema. In the former, 91 per cent of the patients had healed wounds, and were free of symptoms. In the latter, 90 per cent of the patients fell into this category. In contrast to these figures, only 52 per cent of the patients that had multiple abscesses, extensive fibrosis or bronchiectasis were free of symptoms. In this group, 3 patients still had open wounds, with residual cavities in the lung and multiple fistulae; 3 others, although healed, still had symptoms; and 6 others had bronchocutaneous fistulae. All of the cases that were complicated by empyema alone, were of short duration, and the excellent final results in this group were no doubt due to early rupture of the abscess and free drainage into the pleural cavity.

CONCLUSIONS

(1) Early, free drainage of a pulmonary abscess is essential in both the fetid and nonfetid types to avoid the development of complications, the higher operative mortality, and the less satisfactory late results in the complicated cases. For this reason, pulmonary abscess, especially the fetid type, should be accepted as a surgical disease and should be treated under the direction of a surgeon.

(2) The division of cases into "acute" and "chronic," on the basis of time, is confusing. Cases should be classified on the basis of pathology.

(3) A pathologic classification is essential for selecting the proper surgical procedure, and is the most reliable basis for estimating operative risk and late prognosis. It is also important for purposes of comparison between different series of reported cases.

REFERENCES

[1] Wessler, H.: Abscess and Gangrene of the Lungs, Diseases of the Respiratory Tract. Eighth Annual Graduate Fortnight of the New York Academy of Medicine. Philadelphia, Pa., W. B. Saunders Co., p. 295, 1936.

[2] Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung—A Surgical Disease. New York State Jour. Med., **40**, 849, No. 11, 1940.

[3] Graham, E. A.: Pneumectomy with Cautery—A Safer Substitute for the Ordinary Lobectomy in Cases of Chronic Suppuration of the Lung. J.A.M.A., **81**, 1010, 1923. Idem: Cautery Pneumectomy for Chronic Suppuration of the Lung: Report of 20 Cases. Arch. Surg., **10**, 392, 1925.

[4] Lebsche: Cited from Sauerbruch (Sauerbruch, E.: Die Chirurgie der Brustorgane. Berlin, Ed. 3, 1, 900, Julius Springer, 1928).

CONTROL OF MASSIVE ESOPHAGEAL HEMORRHAGE SECONDARY TO LIVER DAMAGE (CIRRHOSIS) BY LIGATION OF THE CORONARY VEIN AND INJECTION OF SODIUM MORRHUATE

EDWIN J. GRACE, M.D.

GRACE CLINIC

BROOKLYN, N. Y.

THE CLINICAL ENTITY of cirrhosis of the liver is relatively constant, in general, but reveals many different causes and many secondary complications. Among the latter, esophageal hemorrhage is frequent and often so severe that death may follow. With our increasing knowledge of liver function, many so-called hopeless cases of liver damage might be helped by using all known data now available for the preservation and restoration of liver function.

The liver is not only the largest but one of the most vital organs in the body, and in spite of its multiplicity of functions the two considered most important are: First, its ability to function with a small amount of normal tissue; and, second, its remarkable capacity to regenerate. Since the causative element in liver damage may in many instances be one not classically accepted or known, every aid should, therefore, be given such patients in spite of the apparently hopeless picture. The multiple functions of the liver make it easy to appreciate the fact that, when this organ is badly damaged, any complication may precipitate a fatality.

Varices, one of the more common complications of the cirrhotic liver, are caused by a greater flow of blood through a vein and by the inability of the muscular and elastic fibers to prevent this increased venous pressure. Since the submucous veins of the esophagus are well supported at the cardia, the formation of varices probably occurs below the cardia and spreads above that point after anastomosis has been completed. The formation of varices above the cardia is undoubtedly more rapid when several large veins cross the cardia. These are probably the first veins in the cardiac region to become enlarged.[4]

Unlike the obstruction of the portal or splenic vein in Banti's disease and splenic anemia, the physiologic changes involved in the occurrence of esophageal varices may have an intrinsic or extrinsic cause. When the portal or splenic vein is obstructed, anastomosis results, in order that the blood from the portal circulation can pass back into the general systemic circulation. Doctor Moersch[7] lists three existing routes through which this may take place, depending upon the site of the obstruction in the splenic or portal vein:

1. At the point of transition between absorbing and protective epithelium.
   (a) Between the coronary vein of the stomach and the intercostal, the azygos, and the diaphragmatic veins

(b) The superior hemorrhoidal with the middle and inferior hemorrhoidal veins

2. Site of embryologic circulation—falciform ligament containing the paraumbilical veins
3. At all situations within the abdomen where the gastro-intestinal tract, its appendages or glands developed from it become retroperitoneal, developmentally, or adherent to the abdominal wall, pathologically.

The first of these is of the most concern, since esophageal varices result from such a communication. This passageway between the coronary vein of the stomach and the azygos, intercostal, and diaphragmatic veins is undoubtedly the most likely to develop, since it is the most direct route between the portal and general systemic circulations. Moreover, as Doctor Moersch points out, the pressure within the portal circulation is increased, when the portal vein is obstructed, with a resultant reversal of the flow of blood through the coronary vein, partly due, no doubt, to the absence of valves. This reversal of the coronary blood flow, in turn, exerts pressure on the esophageal plexus, with the effectual formation of esophageal varices. Anastomosis between the portal and caval circulations takes place within the plexus formed by the branches of the coronary vein after reaching the submucosa of the cardia by way of piercing the muscular coats. Since the veins in the submucosa of the lower esophagus are not very well supported by connective tissue, the increased flow of blood through the vein plus the inability of the venous walls to withstand such pressure, causes varicose veins of the esophagus. Doctor Moersch also points out that the effect of the changing suction in the thoracic cage, due to respiration, must also be considered. Although esophageal varices may involve the greater part of the esophagus, it is usually limited to the lower one-third.

The difficulty of early diagnosis has always been a problem in cases of esophageal varices. Little progress was made in such diagnosis from 1839, when Powers gave the first report of varicose veins of the esophagus to medical literature, until 1925, when Jackson and his associates suggested the value of esophagoscopy. In 1928, Wolf described the roentgenologic technic in diagnosis and, in 1931, Kirklin and Moersch[7] combined roentgenoscopic and esophagoscopic diagnostic technic. The fear of fatal hemorrhage, however, has curtailed the use of esophagoscopy to a certain extent.

For the prevention and control of such hemorrhage, several procedures have been followed at one time or another. Doctor Moersch[7] lists such palliative measures as: "Dietary restrictions; the use of local applications to the varix (diathermy, local compression, *etc.*); injection of solutions into the general circulation to stimulate coagulation; and venesection, as suggested by Drenckhahn, to increase the viscosity of the blood."

Splenectomy has been performed most frequently in an attempt to prevent hemorrhage in the esophageal varices. This evidently decreases by 20 per cent the amount of blood passing through the portal circulation. By splenectomy, also, one source of anastomosis between the portal and caval

circulations is stopped by the severing of the vessels which run through the vasa brevia and which communicate with the esophageal veins. Splenectomy is sometimes followed by an omentopexy with the Talma-Morison operation to divert blood from the direction of the esophageal varices by means of an anastomosis between the portal and systemic circulations. The Eck fistula has also been recommended as one of the simplest methods of remedying varices.[5]

The ligation of the coronary vein to prevent hemorrhage in esophageal varices was first advocated and successfully tried in 1929 by Rowntree, Walters, and McIndoe. Such ligation was carried out to interrupt the flow of blood to the collateral veins in the lower end of the esophagus. This procedure, however, did not preclude all possibility of hemorrhage. In 1933, Kegaries[3] recommended the ligation of the vasa brevia in addition to ligation of the coronary vein in order to prevent the formation of such varices when splenectomy was considered too dangerous. He suggested that mass ligation was possible and that relief from hemorrhage could be obtained by sectioning these veins in the course of splenectomy.

Although injections directly into the esophageal veins have been used as early as 1914, when normal horse serum was recommended by Jackson, Tucker, Clerf, Lukens, and Moore[2] for hemorrhage from ruptured varicose veins of the esophagus, it was not until 1933 that injections of a non-irritating yet sclerosing solution into the periesophageal or paraesophageal plexus (a method similar to that of obliterating varicose veins of the leg) at the same time as ligation of the coronary vein was suggested by Walters.[10] About the same time, Moersch and Pemberton[6] suggested the possibility of injecting a sclerosing solution into the esophageal varices by means of an esophagoscope. Failure to produce esophageal veins in dogs in order to experiment with this new method before attempting such treatment upon the human patient caused the idea to be unadaptable for the moment. In 1939 this method of treatment, the solution being quinine-uretan, was successfully carried out upon the human patient by Frenckner and Crafoord,[1] of Stockholm. Moersch immediately duplicated the procedure with, thus far, successful results.

Nothing is more unpredictable than the favorable clinical results often obtained in spite of extensive liver damage. Modern therapeutic aid has further equipped the clinician. The case here reported represents one of advanced liver damage with ascites and esophageal varices; the latter ruptured, producing severe massive hemorrhage. An apparent hepatic fatality was avoided and the patient restored to improved health by combining ligation of the coronary vein with injection of sodium morrhuate. The patient is now enjoying good health and has no complaints; the hemoglobin is 96 per cent; no ascites are present. A splenectomy was not undertaken on this patient because it was doubted whether such an operation with its definitely higher operative mortality should always be undertaken.

Case Report.—R. T., white, male, age 53, was admitted to the Kings County Hospital in October, 1938, with a classical clinical picture of cirrhosis of the liver; the

patient was cachectic, pale, and cadaverous-looking, with a large abdomen containing fluid and considerable weight-loss. He had had a massive acute hemorrhage from a ruptured esophageal varix. The past history showed that this was his fourth hospital admission for apparently the same complaint, and that he had been constantly subjected,

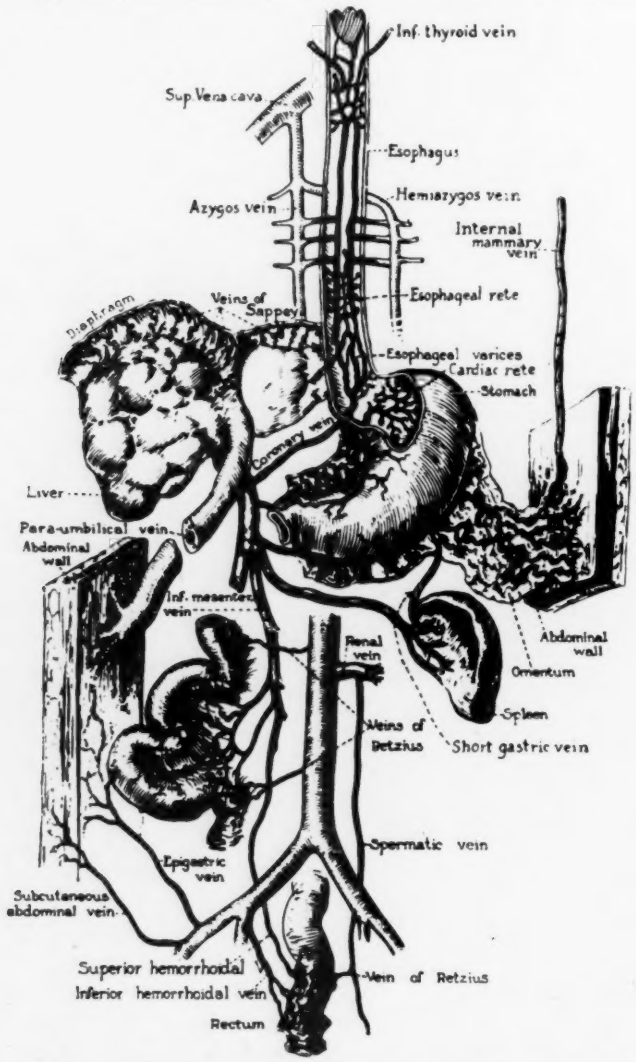


FIG. 1.—Diagrammatic study of the portal circulation with hepatic obstruction showing the anatomic reason for operative procedures described, i.e., ligation of veins of Sappey, ligation and injection with sclerosive solution (sodium morrhuate) of the coronary vein and omentopexy (Talma operation).

over a period of months, to these massive hemorrhages. The hemoglobin was 24 per cent; red count was 1,200,000. During his preoperative period, it was difficult on many occasions to obtain a pulse and blood pressure reading. Several small blood transfusions were given preoperatively with very slight clinical improvement.

In November a celiotomy was performed under a local anesthetic, and when the peritoneum was opened, a large quantity of ascitic fluid gushed forth. The coronary

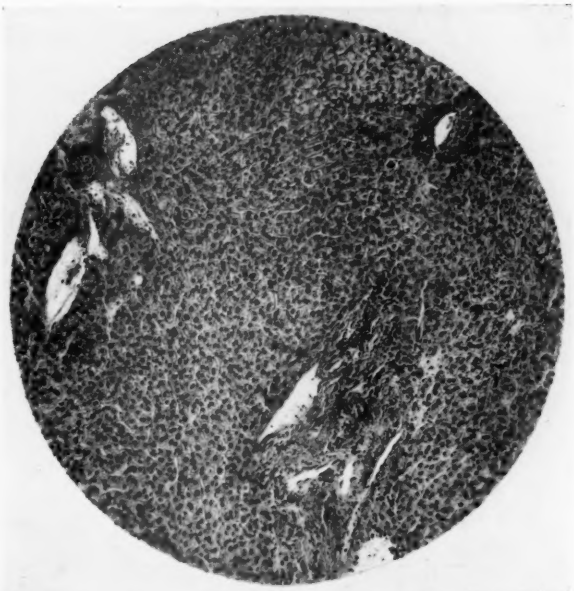
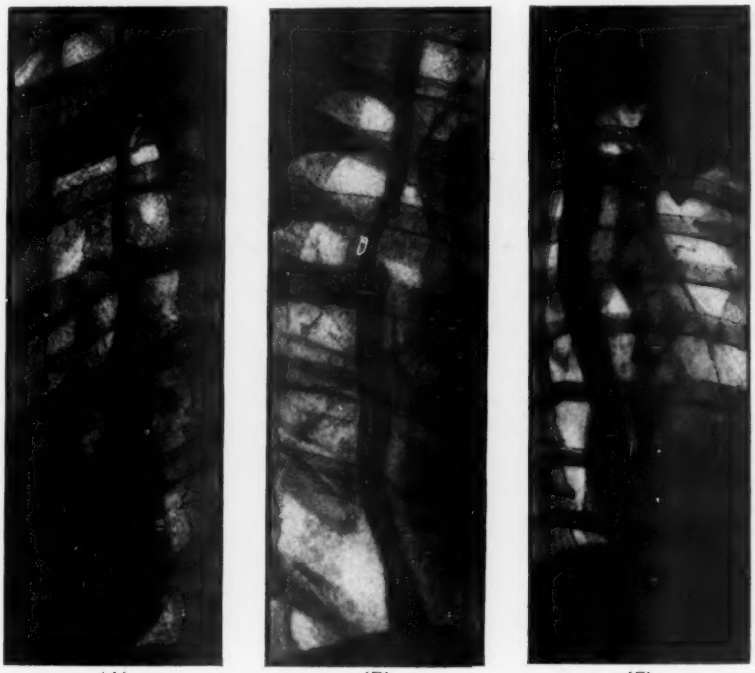


FIG. 2.—Photomicrograph of a specimen of liver removed by biopsy at the time of celiotomy, showing cloudy swelling. (× 159)

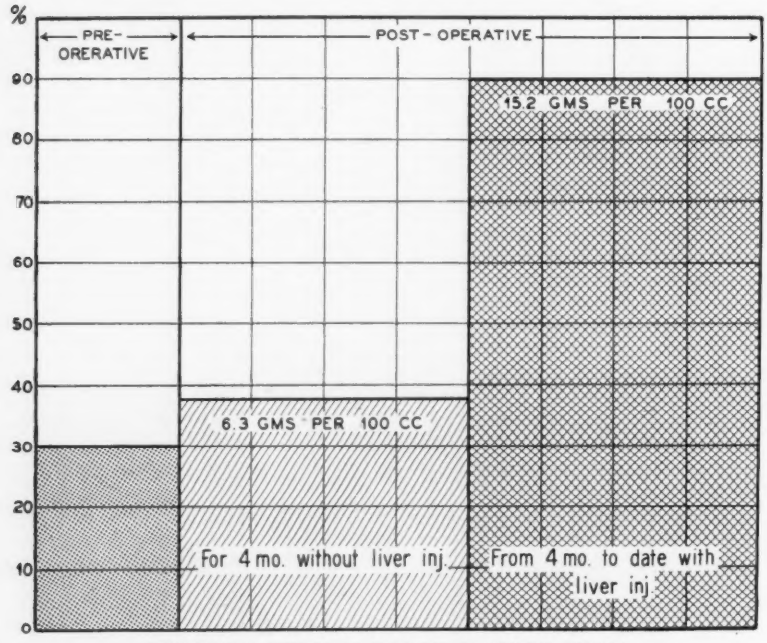


(A) (B) (C)

FIG. 3.—Drawings from esophagograms taken: (A) Preoperatively. (B) Four months postoperatively. (C) Twenty months postoperatively.

vein was found to be enlarged and distended (one centimeter in diameter); it was ligated with black silk, and eight cubic centimeters of sodium morrhuate solution was injected. Three other smaller veins, close to the coronary vein, were also ligated. It is interesting to note that after the above ligations, the veins between the ligature and the liver remained distended, while between the ligature and the esophagus they were collapsed. The veins of Sappey on the undersurface of the diaphragm were also ligated. In view of the above-mentioned collapse of the coronary veins, I did not feel that in this patient the danger of collateral circulation from the spleen was any immediate problem and consequently did not feel that a splenectomy was justifiable. Exploration of this organ showed it to be firmly adherent and bound down,

CHART I



HEMOGLOBLIN STUDIES ON RUPTURED ESOPHAGEAL VARICES
Hemoglobin studies made on June 23, 1941, were 15.2 grams, or 102 per cent.

apparently from an old perisplenitis. The liver was round-edged, pale yellow, and cirrhotic. A biopsy obtained from the liver edge subsequently confirmed the diagnosis of liver damage; however, the specimen was not predominantly fibrotic (Fig. 2). About a month after the first operation, omentopexy was performed (Talma-Morison operation). After this last operation, a mild hemorrhage occurred at two different times, but since then, the patient, when recently seen, appeared to be in excellent health, had had no further hemorrhage and no evidence of ascites. In June, 1940, the hemoglobin had improved from 24 per cent, or four grams, to 80 per cent, or 12 grams. In November, 1940, the hemoglobin had further improved to 96 per cent, or 14.5 grams, according to the photoelectric cell method (Chart I). The liver function test was Grade O, with the serum bilirubin 1.5 mg. per cent.

Roentgenologic studies made preoperatively showed evidence of esophageal varices; when these studies were repeated two months postoperatively, the esophagogram showed alterations indicative of improvement, and similar films taken six months after the ligation and injection of the veins showed a normal esophagus. Although clinical improvement has been marked, roentgenologic studies on August 3, 1939, and at the present

time, show some deviation from the normal, and the general appearance suggests a reversion back to the appearance of the roentgenogram taken preoperatively (Fig. 3). The operation was, of course, directed at a complication of the liver damage (hemorrhage) and only indirectly could benefit accrue to the liver. Although I appreciate fully the limitations of the liver function tests, it is interesting to note that on February 8, and on September 11, 1939, the bromsulfalein showed no dye retention. This, with clinical improvement, is significant. Furthermore, with the control of the massive hemorrhage and the use of all accepted modes of therapy for improving liver damage, such as a diet high in carbohydrates and low in fats; liver extract injections; and especially assimilation of vitamin B, which helps to restore liver function and speeds regeneration of the liver, I feel that these benefits should not be underestimated.

Recent follow-up of this patient, May 15, 1942, finds him in excellent health and working daily running an elevator in a large apartment house.

Liver injections for the past two years have been very irregular, averaging one a month, because of the difficulty the patient has in leaving his work.

REFERENCES

[1] Crafoord, Clarence, and Frenckner, Paul: New Surgical Treatment of Varicose Veins of the Esophagus. Acta oto-laryng., **27**, 422-429, July-August, 1939.

[2] Jackson, Tucker, Clerf, Lukens, and Moore: Hematemesis. Laryngoscope, **24**, 154, 1914.

[3] Kegaries, D. L.: The Venous Plexus of the Esophagus. Proc. Staff Meet., Mayo Clin., **8**, 160, March 15, 1933.

[4] *Idem:* The Venous Plexus of the Esophagus. Surg. Gynec. and Obst., **58**, 46, January, 1934.

[5] McIndoe, A. H.: Vascular Lesions of Portal Cirrhosis. Arch. Path., **5**, 23, January, 1928.

[6] Moersch, H. J.: Treatment of Esophageal Varices by Injection. Proc. Staff Meet., Mayo Clin., **15**, 177-179, March 20, 1940.

[7] *Idem:* Treatment of Esophageal Varices by Injection of a Sclerosing Solution (paper read before the meeting of the American Association for Thoracic Surgery, Cleveland, Ohio, June 6-8, 1940).

[8] Rowntree, L. G., Walters, W., and McIndoe, A. H.: A New Procedure in Management of Cirrhosis of Liver. Proc. Staff Meet., Mayo Clin., **4**, 121, April 17, 1929.

[9] *Idem:* End-results of Tying of the Coronary Vein for Prevention of Hemorrhage from Esophageal Veins. Proc. Staff Meet., Mayo Clin., **4**, 263, September 4, 1929.

[10] Walters, W.: Discussion of reference No. 3.

[11] Walters, W., Rowntree, L. G., and McIndoe, A. H.: Ligation of the Coronary Veins for Bleeding Esophageal Varices. Proc. Staff Meet., Mayo Clin., **4**, 146, May 8, 1929.

SLIDING AND OTHER LARGE BOWEL HERNIAE*

DEVELOPMENT, CLASSIFICATION AND OPERATIVE MANAGEMENT

CLAUDE C. BURTON, M.D.,

AND

CARL BLOTNER, M.D.

VETERANS ADMINISTRATION BUREAU

DAYTON, OHIO

THE MOST serious, the rarest, and the most difficult herniae to repair are the sliding herniae of the large bowel. They are the most serious because the bowel forms a part of the sac and mismanagement may not only result in recurrence as in other inguinal herniae but also in the possible development of a fecal fistula and perhaps general peritonitis with disastrous outcome. Since their incidence is estimated at 1 or 2 per cent but few surgeons have an opportunity to operate upon a sufficient number of sliding herniae to really become proficient in handling them. Usually they occur very unexpectedly, and there are no reliable criteria or pathognomonic signs upon which this type of hernia can be diagnosed preoperatively. Unless the surgeon has familiarized himself with the anatomic development of the large bowel and the various relationships assumed in its embryonal circuit, more particularly the second phase of rotation, he may be baffled as to how to proceed. There are perhaps few situations in the abdomen that require more mature judgment and skill than when one is confronted with a large parasaccular or sacless sliding hernia. Walton[26] defines a sliding hernia "as one in which some portion of the wall is formed by a viscus which in its normal position is only in part covered by pertoneum." Therefore, in its incipiency, a sliding hernia is so totally different from the ordinary saccular type, that it is imperative that the surgeon be vigilant at all times, orienting himself in each step to avoid opening the bowel, mistaking it for the wall of the sac, as was done by Sir Frederick Treves[24], Lawrence[14], and possibly by others, though unreported, with lethal results. This happened to one patient in our series but the bowel was empty, the opening, which was small, was closed immediately, and with the aid of sulfanilamide healed by *per primam*. Also, paralleling in importance, after having recognized the type of hernia present, is the possibility of compromising the circulation by boldly dividing the medial or vascular leaflet. In the large or mature sliding herniae there are problems involving the repair of the fascial and other mural structures equal to, if not surpassing any met with in the various saccular types of hernia. We, therefore, feel that sliding herniae, although admittedly rare, are of sufficient importance to justify redescribing their development, classification and operative management. Incidentally, we

* Published with the permission of the Medical Director, Veterans' Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

are submitting a statistical analysis of 68 cases of large bowel herniae (54 sliding and 14 simple, acquired) in a consecutive series of 2614 repaired inguinal herniae (Table II). This is an incidence of 2 per cent for sliding herniae only.

The literature on saccular herniae, and their repair, is voluminous but, paradoxical as it may seem, it is scanty on herniae of the large bowel including sliding herniae. Moreover, this type of hernia was recognized by Galen during the Hellenic Renaissance in Rome, in the second century A. D. Other early writers include Rousselus,[28] in 1559, Geiger,[28] in 1631, Spigelius,[28] in 1645, and Treves,[24] in 1887. In recent years Garnett[5] (1909), and Bevan[4] (1930), have made significant contributions which seem to us to portray vividly, and simply, the embryologic evolution, classification, and repair of sliding herniae. In the present communication we have drawn freely from these authors.

TABLE I

TYPES OF SLIDING HERNIAE FOUND

Intrasaccular	27
Parasaccular	26
Sacless	1

TABLE II

OPERATIVE FINDINGS IN 68 HERNIAE OF LARGE INTESTINE

	Sliding Type			Simple, Acquired Type		
Structures Involved	Lt.	Rt.	Bilat.	Lt.	Rt.	Bilat.
Sigmoid	28			8		
Cecum		12			2	
Cecum, ileum and ascending colon		3				
Sigmoid and descending colon	2					
Sigmoid and bladder	1					
Cecum, ileum and appendix		3				
Cecum and appendix		1			1	
Cecum, ileum and omentum					1	
Sigmoid and omentum				1		
Cecum and omentum					1	
Sigmoid and cecum			2			
Total	31	19	2*	9	5	0
Total		54*			14	

* Two patients with bilateral herniae counted as four herniae.

Development and Classification.—Carnett classified herniae of the large intestine into the congenital and acquired types. In the congenital type is included those herniae with complete, preformed sacs. The acquired type is subdivided into (a) simple acquired, in which the herniated large bowel is entirely covered by peritoneum and the sac does not form a part of the wall (Fig. 1a); and (b) the sliding hernia in which a portion of the sac is fused with and forms a part of the wall of the herniated large bowel. Sliding hernia may be of the intrasaccular variety (Fig. 1b) with complete sac, or the parasaccular (extrasaccular) variety, with incomplete sac, depending upon the extent of the peritoneal or serous covering of the herniated large bowel (Fig. 1c). A sacless variety occurs but is rare (Fig. 2).

Anomalies in the embryologic development of the small and large bowel are of practical importance to the diagnostician, the roentgenologist and especially the surgeon. Since a sliding hernia is frequently a manifestation of

one of these anomalies, it is essential that the surgeon understand the bizarre conditions that may occur in the rotation of the intestine. It is conceivable to have abnormal disposition of the intestinal loops due to (a) incomplete or nonrotation; and (b) reversed rotation or to deficient fixation. Usually the cecum is completely surrounded by peritoneum except in its attachment to the

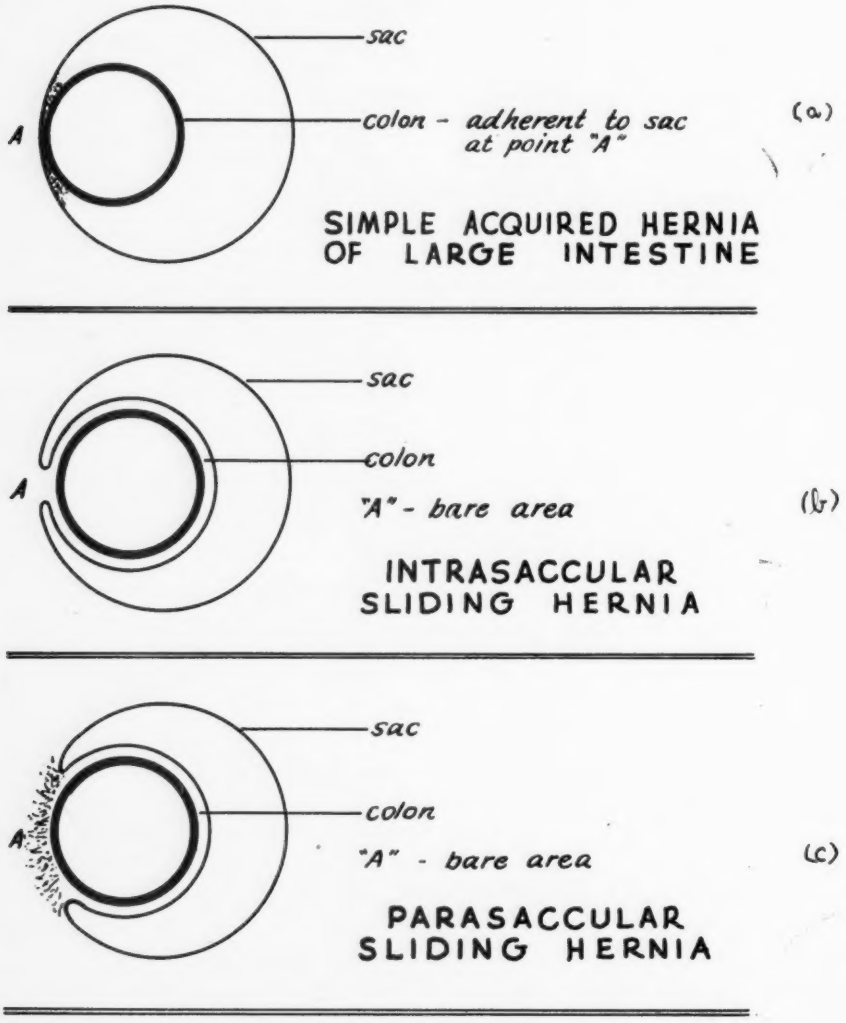


FIG. 1.—Types of Herniae of Large Intestine

colon and does not possess a mesocolon. However, it may have a mesocecum. Rarely a retroperitoneal cecum may escape by the process of sliding through the internal abdominal ring without its serous covering, thus constituting a sacless sliding hernia.

The sigmoid, which is derived from the hindgut, has been, from the very beginning, an extraserous (termed extraperitoneal by others) structure and has not been involved in the process of rotation. Consequently, in sliding herniae of this structure there is usually a comparatively wide aperitoneal, or bare area, and such herniae are more likely to be of the parasaccular type.

Since the serous (peritoneal) covering of the right and left colon may vary widely, as has already been pointed out in their development, the type of sliding hernia encountered will likewise vary accordingly. Another factor to be reckoned with is the intraluminal and static pressure of the bowel forming the wall of the sac which, if continued for long, may convert an intrasaccular type into a parasaccular one.

The next step in the progress of a sliding hernia is its descent into the inguinal canal. As the peritoneum slides over the underlying connective tissue, the attached oncoming bowel, possibly aided by traction of an unusually long mesentery or by the pull from the hernial sac, appears at the internal abdominal ring. If the ring is dilated, and a preformed sac is present, there should be little resistance to the escape of the hernia into the canal. However, if the sphincter at the internal ring is preserved, it may require wedging and pistoning by the hernial mass before the shutter action is impaired and ring stretched sufficiently to permit egress of the sliding hernia into the canal, thus manifesting itself clinically as an indirect type. In the event the internal ring holds, then the next most vulnerable area is the inguinal triangle. Here, again, if there is congenital weakness of the muscle component of the internal oblique it would leave the fascia transversalis as the only barrier to the formation of a mature hernia. Usually the fascia transversalis yields to the intermittent intra-abdominal tension and sooner or later becomes weakened. As the pouching increases in size the fascia transversalis becomes more attenuated until there is marked bulging through the floor of the canal. In this instance the sliding hernia would assume the direct type. Where there is generalized mural weakness with loss of obliquity of the canal in the presence of a coexisting congenital sac, the sliding hernia would have the appearance of the combined or indirect-direct type. Therefore, in dealing with inguinal herniae it is easy to see how,

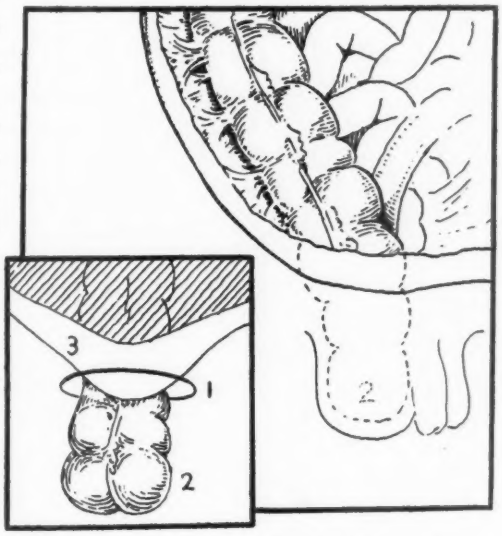


FIG. 2—Sacless Sliding Hernia of Cecum: 1—Internal ring, 2—Cecum without peritoneum, 3—Peritoneum.

in a few brief moments of dissection, one's attitude can be suddenly changed from one of complacency to consternation.

Perhaps the classification of sliding hernia that is most generally used, and a practical one, is that of Bevan[4]. It differs from Carnett's in that it is mainly anatomic rather than embryogenic. Greater stress is placed on the saccular contents than on the extent of the peritoneal investment. Bevan refers to three different forms of sliding hernia: (a) Cecum and ascending colon; (b) descending colon and sigmoid; and (c) the bladder. However, sliding hernia may involve the cecum alone, the cecum and appendix, or the sigmoid alone. At times the mobility of the cecum may be so great as to enable it to assume divers positions within the abdomen. Therefore, it is possible to have the cecum in umbilical, left inguinal, and femoral herniae (Hildebrand[11], and Gibbons[8]). Fortunately, these abnormalities in disposition are rare. The attachment of the peritoneal sac to the bladder for a distance of one to two inches is by no means unusual, occurring in 63 per cent of inguinal herniae in our series. Before diagnosing a sliding hernia of the bladder one should make sure that the bladder wall actually prolapses into the canal and constitutes the primary bulging and not simply auxiliary to the hernial mass. Of course, a concomitant bladder attachment is more frequent in direct or bilocular herniae.

Diagnosis.—A diagnosis of sliding hernia preoperatively cannot be unequivocally established. However, there are certain criteria which cause one to suspect such a hernia. Frequently the patient will relate that he has had a hernia for some time, perhaps several years, and that it has been getting progressively larger. In our series there were a few exceptions to this rule in which the herniae were of a few months duration and had attained considerable size during that brief period. The patient may state that he is unable to wear a truss because of the increased pain during its use. On examination, a dilated external, as well as internal, ring is usually found. Owing to the compromise of function of muscular and fascial structures, there is loss of obliquity of the inguinal canal. A large hernial mass frequently extends down into the scrotum and contains bowel. An attempt to completely reduce this mass may be unsuccessful and give rise to pain. A barium enema may reveal a loop of large bowel in the hernial sac. In the congenital type, traction on the testis after the hernia is reduced causes the hernia to reappear.

Repair of the Sac.—The first prerequisite in the repair of herniae of the large bowel, the vast majority of which are sliding, is the total obliteration of the sac, with the best possible closure of the stump. When dealing with a simple, acquired hernia of the large intestine the adhesions between the bowel and sac are divided and the intestine returned to the abdominal cavity. The sac may then be freed and closed, usually by torsioning an suprasaccular ligation, as described by Russell[23], which is routinely done in all of our inguinal hernia repairs.

On the other hand, when dealing with a sliding hernia, the closure of the sac is a more difficult undertaking. The bowel forming a part of the sac wall

must first be mobilized in order to permit its reduction and proper closure of the sac. To accomplish this safely one must have a thorough knowledge of the blood supply to the involved intestine (Fig. 3). The freeing of the bowel without consideration of this factor may lead to circulatory impairment and gangrene of the involved segment. In the formation of a sliding hernia, we must remember that as the bowel moves or glides down through the internal ring the nutrient vessels are carried along with it. From a study of the anatomy of the large bowel we learn that the blood supply to the colon (except the rectum) enters by way of the medial leaflet of the mesocolon, a fact long recognized, and made use of practically, in successful colon surgery. The lateral leaflet of the mesocolon is avascular. Consequently, in freeing a loop

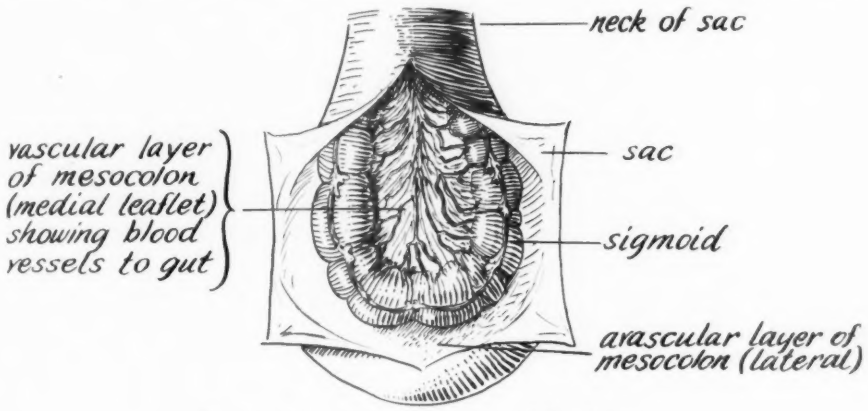


FIG. 3.—Blood supply to intestine in sliding hernia.

of large bowel in a sliding hernia, the lateral leaflet of the mesocolon may be cut with impunity but the medial leaflet must not be disturbed.

In our routine repair of direct, bilocular and other inguinal herniae, we follow Hoguet's[12] technic of freeing the sac and all variants of the sac are indirectalized. The sac is dissected from the bladder whenever such dissection is necessary for adequate high ligation. Our statistics reveal that such a dissection of bladder from the sac was necessary in 63 per cent of inguinal herniae repaired exclusive of sliding herniae. In sliding herniae such a dissection was necessary in but 20 per cent of those repaired.

Several methods of handling the sac in the repair of sliding herniae can be found in the literature. That of Bevan (Fig. 4) may be considered as being perhaps the most satisfactory, especially for the parasaccular type. He incises the avascular or lateral fold of peritoneum of the mesocolon, thereby freeing the bowel. He then raises the bowel and unites the peritoneal flaps behind it but perpendicular to the incision which in effect lengthens the mesocolon permitting reduction of the large intestine forming the wall and at the same time covering the raw surface. This is the same principle which has been employed by Heineke[10], Mikulicz[19], and W. J. Mayo[17], in correcting stenoses of hollow viscera, particularly at the pylorus. After having replaced

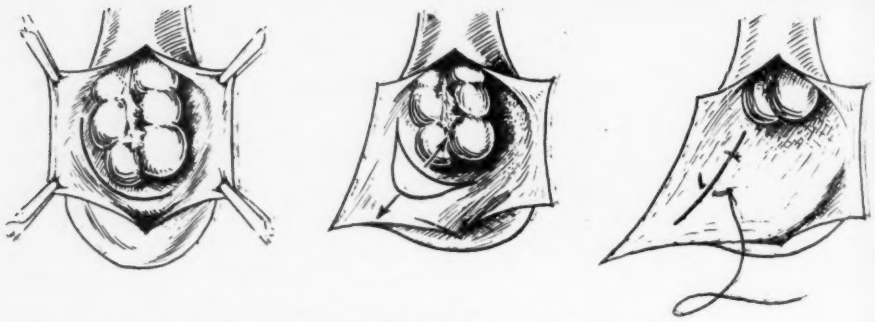


FIG. 4.—Bevan's method of sac closure in sliding hernia.

the bowel in the abdomen the sac is invaginated by means of a series of purse-string sutures, which forms a tampon or cushion at the internal ring.

Carnett describes a method employed by Morris[21], van Heuverswyn[25], Gouillard,[9] and others (Fig. 5). The sac is incised on either side of the reflection of the peritoneum a short distance from its junction with the intestine, thereby producing a free flap of peritoneum on either side of the bowel. Each flap is then turned back behind the bowel so as to cover the bare area and the two are sutured together in this position forming a sort of mesocolon.

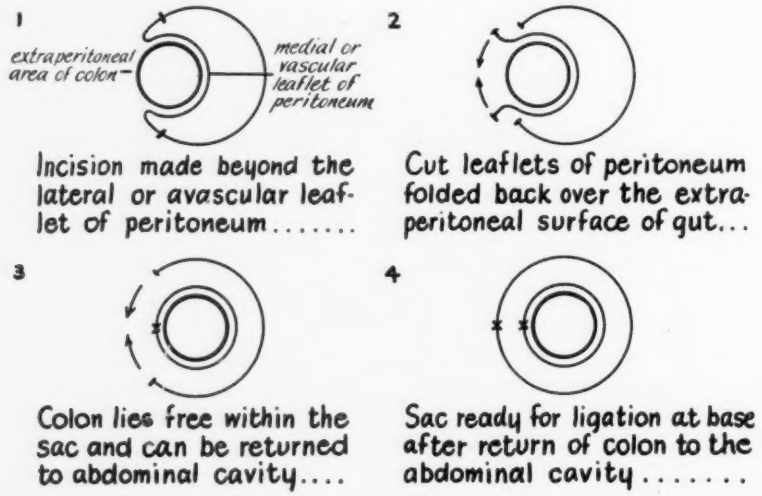


FIG. 5.—One method of handling sac in sliding hernia (view looking down into the sac).

In the suturing of these flaps care should be exercised to avoid traumatizing the vessels in the medial leaflet. The cut margins of the sac are then brought together and sutured, so that the intestine now lies completely within the sac. The bowel is then pushed into the abdominal cavity and the neck of the sac is closed. If used with caution this method is of value especially in cecal herniae.

Another method of obliterating the sac is that of Berger[3] which differs from Carnett's in that the uncut peritoneum on either side is united behind

the bowel, thus forming a mesocolon covering the denuded area. The stump of the sac is then transfixed beneath the internal oblique and transversalis muscles, as originally described by Ball[2] (1884), and subsequently by Kocher[13] (1892).

In some instances in our series of sliding herniae, we have deviated somewhat from the previous methods in that a fish-mouth-type of closure of the sac has been used (Fig. 6). This method consists in overlapping that half of the sac which does not contain bowel over the other half which contains the freed and reduced sliding loop of intestine. Therefore, in this arrangement, the anterior surface of the bowel is covered by a double layer of peritoneum.

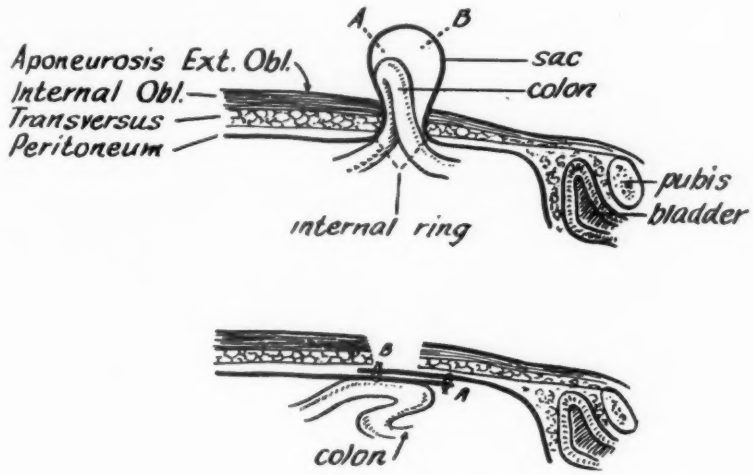


FIG. 6.—Fish-mouth closure of sac in sliding hernia.

Repair of the Wall.—Since herniae of the large intestine are usually of good size, often with concomitant marked structural weaknesses at the internal ring and the floor of the canal, we consider the repair of the wall in such herniae of utmost importance. In the majority of these herniae, particularly the intrasaccular and simple acquired types occurring for the first time, we have employed the method of Ramos and Burton.[22] This method differs from the original Andrews[1] procedure in the placing of the cord extra-aponeurotically instead of interaponeurotically, and the careful denudation of the transversalis fascia of any muscule fibers. It is an all-fascia closure, obtained by suturing the fascia transversalis to the recurved edge of the inguinal ligament, with imbrication of the aponeurotic flaps of the external oblique muscle. This method is based upon principles which are physiologically and histologically sound. It may be used with considerable satisfaction in those instances where there is no attenuation or absence of the fascia transversalis and no structural weakness of the inguinal ligament. In our series, 50 primary herniae of the large bowel were repaired by this

method. Twenty-five of these were followed for an average of 34 months, with three recurrences (Table III).

TABLE III

FOLLOW-UP STUDY AT YEARLY INTERVALS OF 43 SLIDING HERNIAE

Follow-Up—in Months	Number of Cases	Recurrences
72 to 84	5	0
60 to 72	2	0
48 to 60	4	0
36 to 48	5	1
24 to 36	5	0
12 to 24	22	2
Average months 34	Total 43	3, or 6.9%
Exam. by physicians	27	2
Direct correspondence	16	1

TABLE IV

TYPES OF REPAIR—NUMBER FOLLOWED AND RECURRENCES OF HERNIAE OF LARGE INTESTINE

	Number of Operations	No. Followed	Recurrences
Triplicate fascial closure	50	25	3
Fascial sutures (Gallie[7] and McArthur[18])	14	14	0
Cooper's ligament	2	2	0
Pedicled-fascial graft	2	2	0

In primary herniae of the large intestine, with attenuation of the fascia transversalis, we employ a fascial suture according to the method of Gallie,[7] or McArthur.[18] Fourteen herniae have been repaired by this method, with no recurrences.

In recurrent sliding herniae the mural repair is a more formidable undertaking. Usually careful evaluation of the structures will reveal marked thinning or absence of the fascia transversalis, which requires replacement by like tissue without tension if reasonable success is to be expected. In the two cases encountered in our series we elevated a pedicled-graft of fascia lata, passing it through the femoral canal and then fanning it out and uniting it to the lateral edge of the rectus sheath. This is a modification of Wangensteen's[27] method. Those herniae recurring a second or third time should also be repaired by this method. The two cases in which this method was used have been followed two years, without recurrence.

Where there is fragmentation or inadequacy of Poupart's ligament but with strong and retentive fascia transversalis, termed endo-abdominal fascia by Dickson,[6] Cooper's ligament should be used for anchorage in lieu of Poupart's, as originally described by Lotheissen[15] (1898). Follow-up examinations in the two cases in which this type of repair has been used reveal the most resistant walls of any repairs we have used. In fact, we have been so favorably impressed that we are employing this technic in an increasing number of cases, especially in those having marked laxity of the floor.

HERNIOCELIOTOMY

Some of the more adventurous surgeons have introduced a celiotomy in addition to the usual methods of obliterating the sac and repair of the wall. Their contention is that the better exposure permits of greater dissection of the dislocated loop, enabling reduction of the bowel to its former position

and then fixing it in this position by some type of colopexy. A second consideration pointed out by those favoring this method is that the bowel is suspended and does not rest on the internal abdominal ring as in the usual hernial repair. Theoretically the hernioceliotomy may possess certain advantages over the routine plastic procedures that are generally employed, but the complications which may obtain by introducing another incision and the consequent trauma outweigh the theoretic advantages. We, therefore, believe as does Bevan, who states: "It is very desirable not to complicate the operation with a laparotomy." Moreover, with a recurrence rate of only 6.9% in our series, we are reluctant to change our basic ideas in dealing with sliding herniae until greater success has been shown by other methods than is apparent in the statistics so far reported. In those massive herniae in which the fascial floor no longer offers any resistance to the egress of the bulging mass, and perhaps the type ideally suited for a celiotomy, we feel that the retentive structure can be replaced successfully by using a pedicled-graft of the iliotibial tract of fascia lata.

In deference to those surgeons who are exponents of hernioceliotomy, reference is made to Morestin's[20] method. After disposing of the sac, as does Berger,[3] he makes another incision through the abdomen, withdraws the bowel and sutures the base of the newly formed mesocolon to the iliac fascia. The inguinal repair is completed in the usual manner. MacKid[16] uses the bi-incisional technic, replacing the sliding loop of bowel and fixing it there. He then corrects the redundant peritoneum by overlapping the lower loop on the upper, which produces a double layer of peritoneum over the front of the bowel. In the repair of the canal, the aponeurotic flaps are imbricated without transposition of the cord and without using the fascia transversalis or internal oblique muscle, or disturbing the relationship of the funicular structures.

SUMMARY

1. The embryologic development, classification and diagnosis of inguinal herniae of the large intestine have been discussed.

2. The operative findings in 68 herniae of the large bowel have been tabulated.

3. Methods of closure of the sac and the complications that arise in mobilizing the bowel have been redescribed.

4. Indications for various technical procedures in the repair of the wall in primary and recurrent sliding herniae have been discussed and evaluated. The types of operations performed in this series have been tabulated.

5. A statistical analysis of 2,614 consecutive inguinal herniae repaired at this hospital reveals that 68 were herniae of the large intestine. Of these, 54 were the sliding type, and 14 the simple, acquired type. The sliding herniae formed two per cent of all those repaired. Forty-three primary sliding herniae, followed for an average of 34 months, showed a recurrence rate of 6.9 per cent.

REFERENCES

1 Andrews, Edward Wyllys: Imbrication or Lap-Joint Method: A Plastic Operation for Hernia. The Chicago Medical Recorder, August, 1895.

2 Ball, C. B.: The Radical Cure of Hernia by Torsion of the Sac. Brit. Med. Jour. **1,** 461, 1884.

3 Berger: Bull. Med., Paris, **20,** 613, 1906.

4 Bevan, Arthur Dean: Sliding Hernias of Ascending Colon and Cecum; the Descending Colon and Sigmoid; and of the Bladder. ANNALS OF SURGERY, **92,** 754, October, 1930.

5 Carnett, John Berton: Inguinal Hernia of the Cecum, ANNALS OF SURGERY, **49,** 491, April, 1909.

6 Dickson, A. R.: Femoral Hernia. Surg., Gynec. and Obst., **63,** 665, 1936.

7 Gallie, W. E., and LeMesurier, A. B.: Living Sutures in the Treatment of Hernia, Canadian Med. Jour. **13,** No. 7, 1923.

8 Gibbon: J. A. M. A., **30,** 1385, 1898.

9 Gouillard et Rafin: Thèse de Lyon, 1897.

10 Heineke: Reported by F. Fronmüller, Operation der Pylortenose, Inaug.—Dissert., (Erlangen), Fürth, 1886, P. 13.

11 Hildebrand: Deut. Zeit. f. Chir., **33,** 82, 1892.

12 Hoguet, J. P.: Direct Inguinal Hernia. ANNALS OF SURGERY, **72,** 671-674, December, 1920.

13 Kocher, T.: Zur Radical Cur der Hernien. Cor. Bl. f. Schweiz, Aerzte, **22,** 561-576, 1892. Also Transl.: ANNALS OF SURGERY, **26,** 505-526, 1892.

14 Lawrence: Treatise on Rupture, 5th Edition, London, 1838.

15 Lotheissen, G.: Radical Operation for Femoral Hernia. Zentralbl f. Chir., **25,** 548, 1898.

16 MacKid, L. S.: Inguinal Hernia: With Special Reference to Sliding Hernia—A New Treatment. The Canadian Med. Assoc. Jour., **34,** 269, March, 1936.

17 Mayo, C. H.: Gastroduodenostomy. Surg., Gynec. & Obst., **38,** 583, 1924.

18 McArthur, L. L.: Autoplastic Suture in Hernia and Other Diastases: Preliminary Report, J. A. M. A., **37,** 1162, 1901. Final Report, J. A. M. A., **43,** 1039, 1904.

19 Mikulicz, J.: Zur Operativen Behandlung des Stenosirenden Magengesch Wures. Arch. f. klin., Chir., **37,** 79, 1888.

20 Morestin: Congr. franc. de chir., 1900.

21 Morris: Lancet, **2,** 979, October 19, 1895.

22 Ramos, Raoul L., and Burton, Claude C.: Inguinal Hernia: Application of Cardinal Principles in the Repair of Inguinal Hernias. Surg., Gynec. & Obst., **69,** 688-693, 1939.

23 Russel, R. Hamilton: Inguinal Herniae: Their Varieties, Mode of Origin and Classification. Brit. Jour. Surg., **9,** 502, 1922.

24 Treves, Frederick, Sir: Hernia of Cecum. Brit. Med. Jour., **1,** 382, 1887.

25 van Hueverswyn: Jour. des Sci. Med., de Lille, **16,** 121, February 10, 1893.

26 Walton, Albert J.: Extrasaccular Hernia. ANNALS OF SURGERY, **47,** 86-105, 1913.

27 Wangensteen, O. H.: Repair of Recurrent and Difficult Hernias and Other Large Defects of the Abdominal Wall: Employing Iliotibial Tract of Fascia Lata as a Pedicle Flap. Surg., Gynec. & Obst., **59,** 766, 1934.

28 Watson, L. F.: Hernia, 2nd Edition, St. Louis, C. V. Mosby Co., 1938.

SPIGELIAN HERNIA

SPONTANEOUS LATERAL VENTRAL HERNIA THROUGH THE SEMILUNAR LINE

LOUIS P. RIVER, M.D.
OAK PARK, ILL.

FROM THE DEPARTMENT OF SURGERY, LOYOLA UNIVERSITY MEDICAL SCHOOL, CHICAGO, ILL.

THE LINEA SEMILUNARIS (Spigelii) is the line of transition between the muscle bundles and aponeurosis of the transversus abdominis muscle. Its convexity is outward; it lies farthest from the rectus margin at the level of the umbilicus, decreasingly distant from there downwards. It does not coincide with the line of transition in the internal oblique, which, unless extreme muscular aplasia exists, follows the rectus margin quite closely. Lateral to the rectus and medial to the muscular portion of the transversus, the aponeurosis of the latter is almost inseparably adherent to the posterior surface of the internal oblique muscle. However, and particularly at the point where the aponeurosis passes forward and fuses with that of the internal oblique, there are found numerous, up to 3–4 mm., irregular slit-like defects. It is here, at the junction of the semilunar and semicircular (Douglas') lines that almost all reported lateral ventral herniae have been found, i.e., lateral to the rectus, medial to the muscular portion of the transversus, and at or below the fold of Douglas, which usually forms part of the ring. It is possible that these defects are widened by the outward pull of the accessory slip of internal oblique described by Chouke[10] as occurring in a majority of individuals. They certainly vary with the major variations in Douglas's fold, described by him, and by McVay and Anson.[10a]

Many authors, following the anatomic studies of Ferrand,[13] Thevenot and Gabourd,[25] and Beaudoin,[6] have ascribed great etiologic significance to a lateral branch of the deep inferior epigastric artery traversing one of these defects. Koljubakin,[18] alone amongst others who have searched for them, has found them with frequency, anatomically, or reported them as emerging through the hernial ring.[12, 17, 19, 23] There is a relatively constant small lateral branch of this artery running upwards and outwards behind the rectus. It is not constant in the point of passage into or through the transversus; it lies in front of the transversalis fascia hence not piercing that important structure. The defects commonly seen are larger than any through which I have seen this artery pass. The artery probably has slight etiologic importance. Properitoneal lipoma preceding the hernia has been frequently described. It may be noted here that the relatively uncommon direct inguinal hernia with a small tight neck, a prehernial lipoma and a marked tendency to strangulation is a hernia through Spigel's line, emerging through, rather than below, the transversus aponeurosis. The frequency with which semilunar line hernia has occurred in the subjects of other herniae; and

with straining, in subjects with weakened abdominal walls impels one to consider congenital susceptibility, muscular atrophy, pregnancy, obesity, emaciation, chronic cough and severe muscular effort as immediate predisposing factors.

Although spontaneous lateral ventral hernia had long been known, Klinkosch was the first, in 1864, to locate it specifically in Spigel's line. Since that time, there have been many anatomicosurgical reviews of collected cases. [1, 2, 4, 5, 16, 18, 19, 20, 26] Allowing for duplications, and excluding cases unquestionably not spigelian hernia, there have been 99 true spontaneous cases reviewed; I have been able to find reports of 13 additional,[8, 9, 12, 15, 17, 22, 23, 24, 25] making, with the four here to be reported, 116. Only four cases are reported in the American literature.[11, 15, 16, 28] There is one collective review,[16] and one good textbook account.[27] In the 20 years since Holloway,[16] and Auge and Simon[4] reviewed a total of 58 cases, one case has been reported in America, 53 elsewhere.

The hernial ring is lateral to the rectus margin, seldom above Douglas' fold, lying at the juncture of the middle and outer thirds of a line from anterior superior spine to umbilicus. It may be from 0.5 cm. to 2 cm. in diameter, seldom larger. Bonetto[8] described one of 4 cm. The edges are a sharply defined defect in the transversus aponeurosis. The upper, and upper-lateral margins are made crescentic and sharper by the edge of Douglas' fold.

The sac is formed of peritoneum and occasionally fibers of the transversalis fascia. Its thickness seems to depend on duration. It penetrates the transversus aponeurosis and the internal oblique muscle or aponeurosis. It may then spread beneath the external oblique aponeurosis, seldom perforating this relatively free layer. Hence, the hernia is commonly interparietal, the "masked" hernia of Macready. The sac may be from pea to orange size. Enormous herniae have been described by Jaboulay,[4] Le Marie and Pitschke,[4] Lobello,[19] and Fournier.[14] Brandtner,[9] Berger,[7] and Nystrom[21] reported bilateral spontaneous semilunar herniae. Coley[11] felt that the semilunar hernia he reported was traumatic in origin, but the typical location and character of the ring, the interparietal sac, and the presence of indirect and direct inguinal herniae suggest that the indicated fall was only incidental. Steimker[4] observed a similar case at autopsy. Golderger and Panebianco[15] described a large hernia in the semilunar line above the umbilicus, associated with epigastric, para-epigastric and umbilical herniae. Scopinaro[24] examined in life and at necropsy a six-day-old infant with congenital strangulated spigelian hernia in which a fecal fistula formed. Two brothers had died at seven days and six weeks of age, respectively, of identical conditions. The father had bilateral inguinal herniae.

Adhesions, with irreducibility, incarceration, and strangulation have been commonest in the larger herniae; small intestines and omentum are the common contents of the sac, colon rarely. In Case 1 of this report a sliding spigelian hernia of the sigmoid was found. Beaudoin described a multi-

locular, and apparently sliding, spigelian hernia containing cecum, appendix, small intestine, and omentum. The sac in Teales' case contained colon.[16] I was able to find only these two cases resembling Cases 1 and 2 of this report.

Spigelian herniae, even of fair size, may be symptomless. They may cause mild to severe, localized or more diffuse neuralgic pain, aggravated by straining and relieved by spontaneous or manual reduction of the hernia.

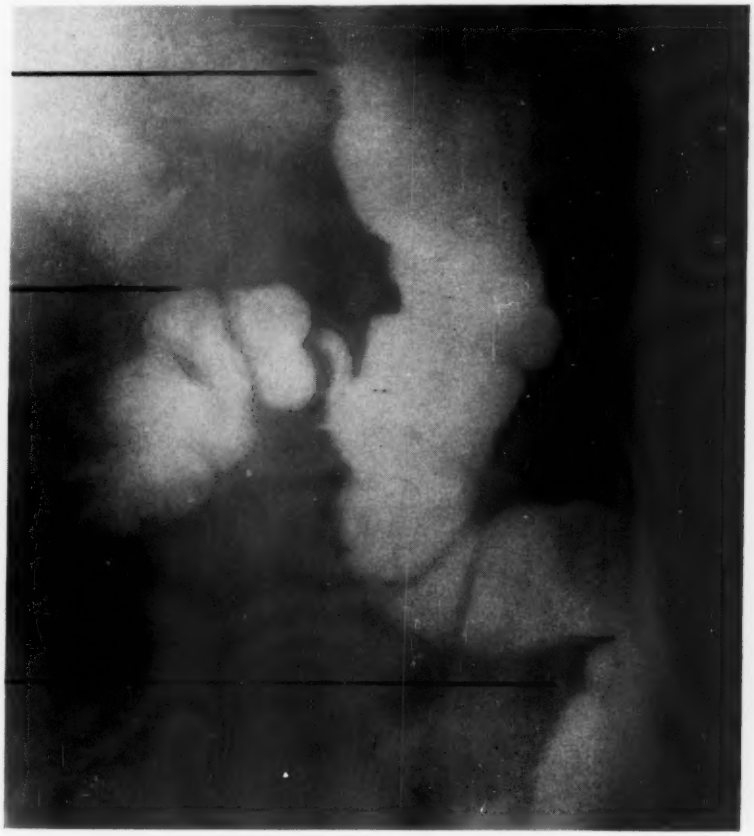


FIG. 1.—Case 1: Lateral roentgenogram showing the anterior fixation of the sigmoid loop, postoperatively.

It may be impossible to palpate a small interparietal mass, especially in an obese patient. However, a localized point of tenderness in the abdominal wall, significantly located at the site of the ring, can usually be found. Watson[27] feels this to be best elicited in the erect position. It has seemed to me best demonstrated by palpating along the rectus margin of the supine patient, straining to raise the head and shoulders. This procedure quite definitely excludes visceral tenderness.

The four herniae, upon which this report is based, were spontaneous, interparietal and located at the junction of semilunar and semicircular lines. One was large, multilocular, incarcerated, and strangulated. One was a sliding hernia.

Case 1.—A. W., age 64, a well-developed, well-nourished male, had done strenuous work in his youth; extensive walking during the past 10 years had been with effort because of an arthritic hip. Two years ago (one year after an operation for strangulated right indirect inguinal hernia), he noted a "lump" on the left side, easily reducible by pressure with his left thigh flexed. When it protruded, especially in the morning, he had

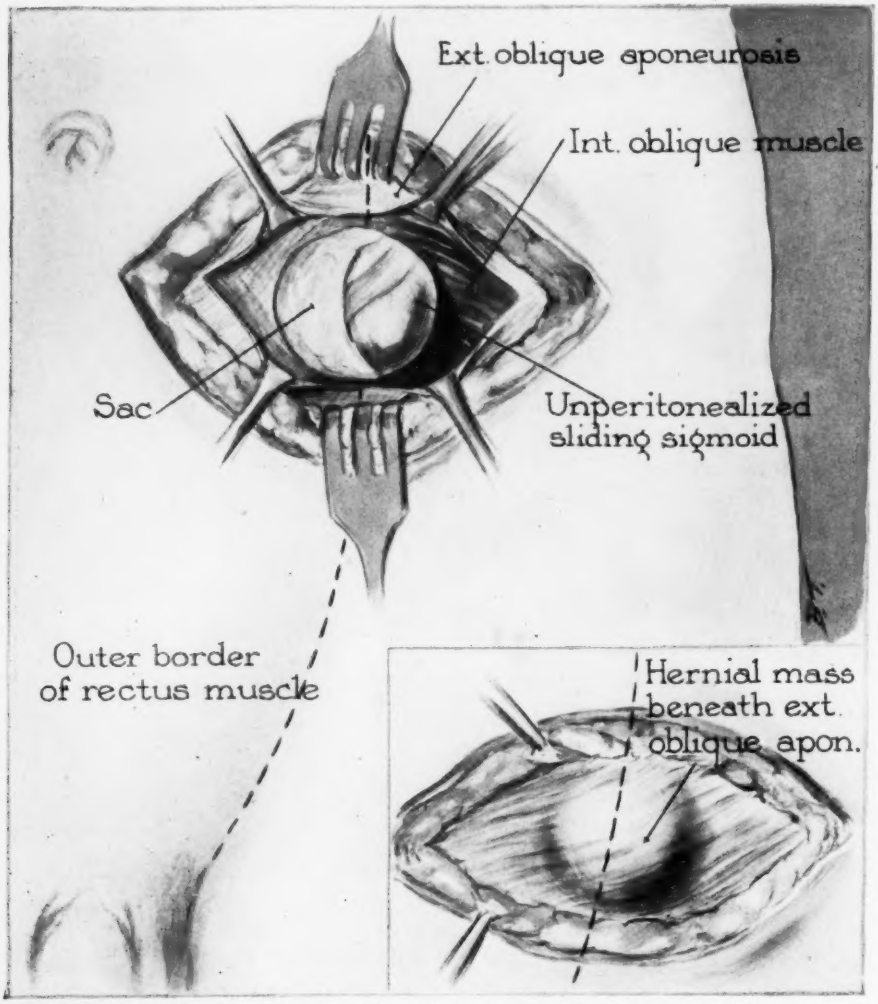


FIG. 2.—Case 1: Showing anatomic relations of the structures involved, as noted at operation.

a dull aching pain and distress from "gas". He blamed his constipation on the lump, stating that if he held it reduced he could pass gas and have his usual morning bowel movement. On three occasions I was unable to palpate the mass or the ring, and he could not produce the lump by straining. On November 1, 1937, he came in saying the lump was then present. An indefinite mass was palpable at the lateral margin of the rectus muscle at the level of the anterior superior spine. It disappeared with a gurgle upon lying down; the area remained moderately tender to deep palpation. No ring was felt. He was admitted to Oak Park Hospital.

Operation.—November 2, 1937: Under novocain block, a transverse incision was made at the level of the anterior superior spine. The ring was about 2 cm. in diameter, and the thickened margin consisted of transversus and internal oblique fibers. When he strained the sac protruded to a height of about 3 cm. beneath the external oblique aponeurosis. The lateral wall of the sac was unperitonized sigmoid colon.

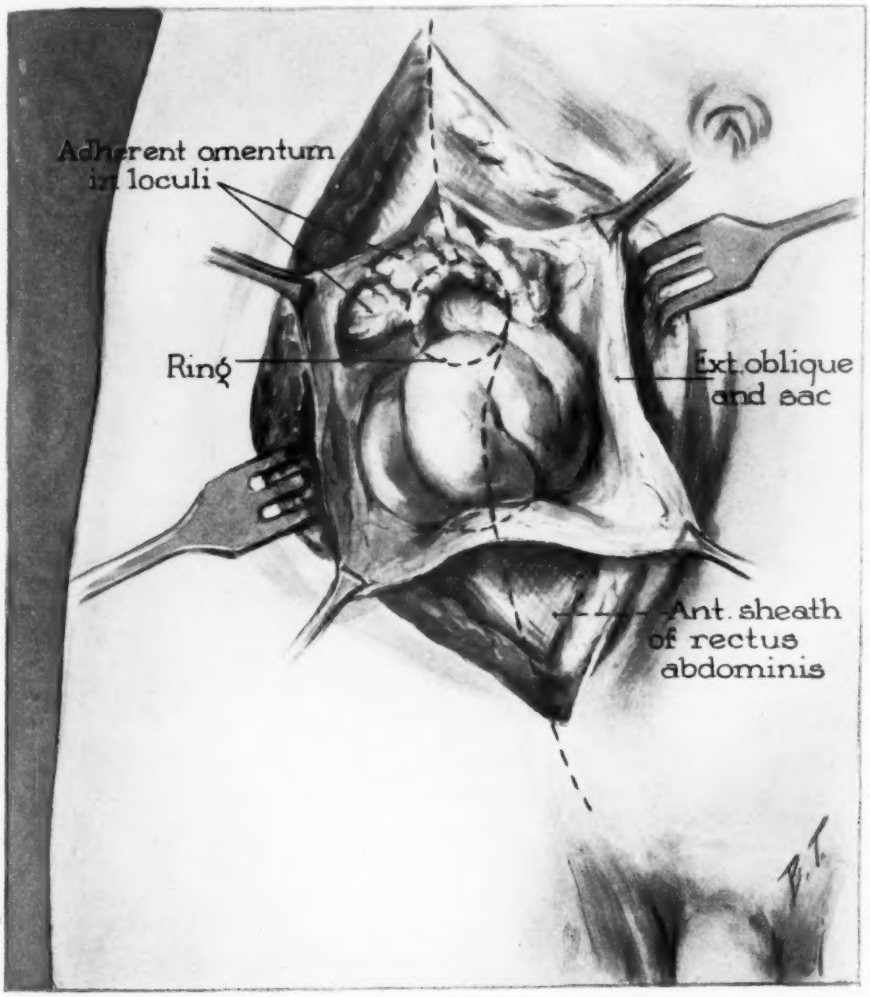


FIG. 3.—Case 2: Showing anatomic relations of the structures involved, as noted at operation.

The incision in the sac was closed, the intestine pushed back, and the peritoneal suture anchored to the lateral angle of the transversus aponeurosis. Closure was by transverse imbrication of the ring margins and simple closure of the external oblique. Recovery uneventful, and he has remained well since.

Case 2.—T. M., a well-developed, well-nourished, somewhat obese male, age 54, was admitted to the Cook County Hospital, August 6, 1939, at 7.30 P.M. He complained of colicky abdominal pain, nausea and vomiting, and obstipation for 48 hours. He had had a hernial mass in the right lower quadrant lateral to an old midrectus scar, slowly increasing in size for 20 years. The abdominal pain seemed to have

originated at that point. Twenty-four hours previously a physician had unsuccessfully attempted reduction. The past history was irrelevant except for appendicectomy in 1914. He had never paid much attention to the hernia.

His abdomen was obese; there was doughy distention, the bowel sounds were infrequent and obstructive in character. There was a large, bulging, irregular, firm, poorly defined mass in the right lower quadrant, lateral to the old scar. The contents of the lower half of this irregular loculated hernia were reducible, of the upper, more tender portion, irreducible. His general condition was good.

Operation.—Under spinal analgesia, an oblique incision over the mass was made through skin and external oblique aponeurosis. Lying between the external and internal obliques, the hernial sac had four walnut-sized loculations surrounding a 2.5 cm. circular defect in the transversus and internal obliques at the junction of semilunar and semicircular lines. The right rectus muscle was thin and atrophic. Extending down to the inguinal region was a loculation of orange-size. This contained reducible loops of undamaged ileum, the four small ones contained adherent incarcerated omentum which was especially adherent to the ring. No defects were palpable at the umbilicus, the right inguinal and femoral rings or the inner surface of the appendicectomy scar. After dissection and resection of the involved omentum, clean definition of the defect, and closure of the peritoneum, the transversus aponeurosis was imbricated transversely with interrupted mattress sutures of silk. The lining of the lower part of the sac was imbricated over this and the external oblique and skin closed. He made an uneventful recovery, and upon examination, February 10, 1941, had no palpable recurrence.

Case 3.—V. A., age 21, single, a well-developed, thin, white female, had had attacks of right-sided pain and soreness for a year. It had been said to be due to appendicitis. The pain disappeared on lying down and had no relation to alimentary rhythm. There was a small tender area, to which she pointed accurately, in the semilunar line just above the level of the anterior superior spine. No mass or ring was felt.

Operation.—January 29, 1939, at Oak Park Hospital: A transverse incision was made. The internal oblique muscle was intact but thin, almost aponeurotic. There was a 1.5 cm. elliptical defect in the transversus aponeurosis, the upper margin being the fold of Douglas, through which the peritoneum bulged. Closure as in Case 1. The appendix was grossly and microscopically normal. Recovery was uneventful, and relief has been complete.

Case 4.—A. S., age 35, married, multiparous, a well-developed, well-nourished white female, had had almost similar pain, also disappearing upon reclining. She had had a supracervical hysterectomy in 1936, and had been told that her residual left-sided pain was due to a slightly cystic left ovary. No mass or ring was felt at the tender spot.

Operation.—August 3, 1939, at West Suburban Hospital: A one centimeter elliptical defect in the transversus and internal oblique was found, slightly above the level of the anterior spine. She has remained free of pain following repair.

SUMMARY

1. The anatomy of the transversus aponeurosis is reviewed, and observations pertinent to the etiology of semilunar line hernia added.

2. Case reports appearing since the last collective review are noted.

3. Constant location, interparietal "masked" character, and apparently frequent strangulation are noted as leading to the belief that the hernia when small and uncomplicated, is possibly frequently overlooked as a cause of pain.

4. Four cases are reported.

BIBLIOGRAPHY

[1] Angeletti, E.: Spigelian Hernia. Review. Arch. Ital. di. Chir., **17,** 38-60, 1927.

[2] Anzilotti, E.; Clinical and Therapeutic Study of Three Cases of Spigelian Hernia. Arch. Ital. di. Chir., **50,** 107-119, 1938.

[3] Apfelthaler, M.: On the Occurrence of Ventral Spigelian Hernia. Zentralbl. f. Chir., **51,** 1680, August 2, 1924.

[4] Augé, A., and Simon, R.: Contribution to the Study of Hernias in the Semilunar Line of Spigel. Rev. de Chir., **59,** 297, 1921.

[5] Barthelémy, M.: Lateral Abdominal Hernias. Bull et. Mem. Soc. de Chir., de Paris, **45,** 1313-1319, October 15, 1919.

[6] Beaudoin, Quoted by Holloway.[16]

[7] Berger. Quoted by Barthelémy,[5] and Holloway.[16]

[8] Bonetto, E., Ventrolateral Hernia of Linea Semilunaris. Rif. Medicale, **54,** 875-877, June, 1938.

[9] Brandtner, C. E.: Case of Traumatic Hernia in the Spigelian Line. Arch. für orthopädische und Unfall-Chirurgic, **33,** 219, 1933.

[10] Chouke, K. S.: The Constitution of the Sheath of the Rectus Muscle. Anat. Record, **61,** 341-348, June, 1938.

[10a] McVay, C. B., and Anson, B. J.: Composition of the Rectus Sheath. Anat. Record, **77,** 213-225, June, 1940.

[11] Coley, W. B.: Interparietal Ventral Hernia at McBurney's Point. ANNALS OF SURGERY, **50,** 246-249, 1909.

[12] Derycke, P.: Contribution to the Study of So-called Hernia of the Zone of Spiegel. Bruxelles Med., **18,** 85, May 1, 1938.

[13] Ferrand: J. A. M. J., Quoted by Thevenot and Gabourd.[26]

[14] Fournier, R.: Hernia of Spigel's Line. Bull. Soc. d'obst. et Gynec., **22,** 695, October, 1933.

[15] Goldberger, H. A., and Panbianco, R. R.: Multiple Hernia. Am. Jour. Surg., **42,** 423, November, 1938.

[16] Holloway, F.: Spontaneous Lateral Hernias. ANNALS OF SURGERY, **75,** 677, June, 1922.

[17] Junet, W.: Hernia of the Semilunar Line of Spiegel. Acta Helvet. Med., **4,** 403, August, 1937.

[18] Koljubakin, S. L.: Hernia of Spigels' Line. Arch. f. klin. Chir., **136,** 739, 1925.

[19] Lobello, F.: Contribution to the Knowledge of Spontaneous Hernia of Spigels' Line. Riforma Med., **50,** 525, April, 1934.

[20] Mackrocki: Quoted by Augé and Simon.[4]

[21] Nyström, G.: Two Cases of Hernia of Linea Spigelii. Acta chir. Scandin., **56,** 92-95, 1923.

[22] Odes, L. A.: Hernia of the True Semicircular Line of Spigel. Vestnik Khir., **44,** 49, 1936.

[23] Papin, F.: Strangulated Hernia of Linea Semilunaris Spigelii. Bordeaux Chir., **7,** 203, April, 1936.

[24] Scopinaro, A. J.: Hernia on Spigels' Semilunar Line in a Newborn. Semana Medicole, **1,** 284, January 24, 1935.

[25] Seara, P.: Anatomico Surg. Study of the Line of Spigelius. Pathology of Spigelian Hernia. Dia. Med., **10,** 972, September 19, 1938.

[26] Thevenot, L., and Gabourd, T.: Spontaneous Hernias of the Semilunar Line of Spigel. Rev. de Chir., Paris, **35,** 568-585, 1907.

[27] Watson, L. F.: Hernia, 2nd ed. p. 370, St. Louis. C. V. Mosby, 1938.

[28] Watson: Hernias of Upper Abdomen. Memphis Med. Monthly, **40,** 461-464, August 1, 1919.

[29] Williamson: Quoted by Holloway.[16]

THE PREPARATION OF NONPYROGENIC INFUSION AND OTHER INTRAVENOUS FLUIDS BY ADSORPTIVE FILTRATION*

REPORT OF 42 MONTHS' TRIAL

Co Tui, M.D., and A. M. Wright, M.D.

New York, N. Y.

FROM THE LABORATORY OF EXPERIMENTAL SURGERY AND THE DEPARTMENT OF SURGERY, NEW YORK UNIVERSITY COLLEGE OF MEDICINE, NEW YORK, N. Y.

Part I—Biologic Aspects

In 1937, there was reported from this laboratory a method of preparing intravenous fluids based on a new principle.[1, 2] As pointed out in that report, it was Siebert's[3, 4] extension of Hort and Penfold's[5] work which laid the foundation for the various methods of preparing intravenous fluids at present practiced in commercial houses and hospitals. Even the method described by Rademaker[6, 7] and the more recent and mechanically ingenious elaboration by Walter,[8, 9, 10] stem from Siebert's original principle. The principle, however, suffers from a critical weakness in the assumption that if the water used in the manufacture of the fluids is pyrogen-free, then the resulting mixture must also be pyrogen-free. But should the water become contaminated, say as a result of failure to clean the still, or should pyrogen be present in the chemicals used in making the solutions, the lack of provision for its removal is a notable disadvantage of the method. It is, in fact, the lack of security behind the old working principle which impels manufacturers to test their intravenous fluids on the rabbit. Unfortunately, not even this precaution has entirely eliminated the occurrence of pyrogenic reactions. As pointed out in a previous communication,[11] this failure is perhaps due to the inadequacy of the test doses in use.

That chemicals occasionally are contaminated with pyrogen has not heretofore entered into consideration. In previous papers[12] we have reported on the pyrogenicity of samples of inulin as they have come fresh from the manufacturer.

Table I shows the effect on test animals of samples of sodium chloride, of dextrose, and of sodium citrate, all "commercially" and "technically pure," which had been made into solutions with nonpyrogenic water and tested immediately after the containers had been opened. The sodium chloride sample was the more reactive of two found pyrogenic in a test of 38 samples, an incidence of slightly more than 7 per cent. The dextrose was the most reactive of four positive samples of 32 tested, an incidence of 14 per cent, and the sodium citrate was the most reactive of four of 18 tested samples, an incidence of over 22 per cent. It may also be mentioned that sodium citrate

* Aided by the Departments of Hospitals and Purchase of the City of New York, N. Y., and a grant from the New York Foundation.

is usually more heavily contaminated than dextrose which, in turn, is usually more heavily contaminated than sodium chloride. Because of the fact that test animals injected with such large doses of sodium citrate are liable to die in cardiac arrest, perhaps as a result of the blood calcium being thrown out of ionization, it is necessary to add an equivalent dose of calcium chloride to the sodium citrate test solution before it is injected.

TABLE I

EFFECT OF VARIOUS COMMERCIAL SOLUTIONS

Exper. No.	Chemicals	Test Dose Gm. Vol. Cc.	Animal Used	Weight Kg.	Temp. Rise Deg. F.	Leukocyte Count Change x 1000
1	Sodium chloride....	.4 50	Rabbit	3	1.2	
2	Sodium chloride ...	12 1500	Dog	15	2.0	—5.7
3	Dextrose.....	7.5 150	Rabbit	3	2.0	
4	Dextrose.....	75 1500	Dog	15	2.4	—9.4
5	Sodium citrate*....	0.28 11.5	Rabbit	3	2.2	
6	Sodium citrate*....	2.8 115	Dog	15	2.8	—8.2

*These dosages were calculated on the bases that the maximum dose of sodium citrate given to a 60-Kg. man is 5.6 Gm. (3 liters of blood), and that the rabbit is approximately three times and the dog six times less sensitive to pyrogen than man. An equivalent amount of nonpyrogenic calcium chloride was added to each sodium citrate solution.

The chemicals in these cases were perhaps contaminated by pyrogen in the process of manufacture. Nonpyrogenic chemicals even in the "dry" state can, however, become pyrogenic on standing under unsterile conditions, as has been found by Smith[13] to be the case with inulin, and by us with dextrose, sodium citrate and heparin. Apparently the better medium the chemical is for bacterial growth, the more easily it becomes pyrogenic. Humidity and a warm environmental temperature seem to accelerate pyrogen production, probably by acceleration of bacterial growth. Among aerial contaminants, Co Tui and Schrift[14, 9] have found *B. proteus vulgaris* and *B. subtilis* fairly prolific pyrogen producers. In the light of these findings, therefore, the assumption that to guard against the occurrence of pyrogenic reactions it is necessary only to keep the water pyrogen-free can be costly as well as unsafe. The problem will perhaps become increasingly acute with the increasing use of blood and plasma transfusions.

The adsorptive filtration method of preparing intravenous fluids differs from the older method in that it is based on the principle of filtering the resulting mixture of chemicals and distilled water through specially prepared compressed asbestos pads proved to remove pyrogen. This method is efficacious not only for infusion fluids but for all crystalloidal intravenous medicaments. With colloidal substances such as sera and gum acacia, however, it is ineffective. The method has been in experimental use on the New York University Surgical Division of the Bellevue Hospital continuously since July, 1937 except for an intermission of 16 months. It is the purpose of the present communication to summarize the results of the experiment as well as to describe the practical method of manufacture which has been evolved.

The practical difficulties encountered in the course of this work may be briefly discussed since they are difficulties which many hospitals and similar institutions which undertake the manufacture of their own infusion fluids will have to face.

1. The first problem was the establishment of the criterion of pyrogenicity. With fluids which are frankly pyrogenic, this is an easy matter since the symptom-complex of the pyrogenic reaction is a familiar one. The difficulty was with fluids containing a borderline dose of pyrogen, which would elicit only a slight temperature rise easily missed in the four-hour routine ward temperature readings but might, when administered in doses two or three times larger than the usual one, give rise to a fairly severe reaction.

It was, therefore, necessary during the first six months of this experimental period to subject the fluids made to animal tests, using the rabbit and the dog, according to the technic described elsewhere.[11] If the fluids were found nonpyrogenic according to the tests, they were then released for ward use. The temperature readings of all patients receiving the fluid were taken hourly for four hours, both during and after the administration of the fluid. In this way transient temperature rises could be detected.

2. The second problem was the elimination of contamination from external sources, such as the rubber tubings, glass connections and needles used in the dispensing set. Originally, the latter were sterilized in the ward instrument sterilizers in common with contaminated pus basins and surgical instruments. The occurrence of four reactions on patients in the first three weeks of this period led to tests of the water from the sterilizer. Ten and 15 cc. samples of this water gave unmistakable reactions in both rabbits and dogs. This result led to the institution of a method of washing, packing and sterilizing the dispensing sets which will be described in a later section of this paper.

3. A third difficulty was the presence of a yellowish-brown coloration in the solutions after they had been autoclaved. This was first thought to be due to caramelization but has been attributed by Krno[15] to the production of levulinic acid.[10] In our preparation two factors were found responsible for the coloration: (a) The asbestos pads originally used were made for the filtration of fluids which did not have to be autoclaved. They imparted some principle to the filtrate which caused the formation of this coloration on heating. The adoption of a pad specially prepared for infusion fluid use has eliminated this factor; and (b) the second factor was overheating of the solution. This occurred when, after sterilization, the autoclave pressure was permitted to sink so fast that the fluid inside the flasks began to boil over. The film of fluid on the inner wall of the neck of the flasks evaporated and the residue became browned, imparting the coloration to the rest of the solution. The remedy to this was obviously to lower the autoclave pressure so slowly that no boiling over took place.

4. The fourth and most serious difficulty was the presence of shreds and particles in the filtrate. While the injection of solutions containing these particles into numerous experimental animals caused no detectable symptoms,

their presence was none the less objectionable. It was because of this difficulty that the use of these fluids was suspended for 16 months in 1938 and the first part of 1939, until a manufacturer could be found who would cooperate in devising means of eliminating these particles. Jena filters, Chamberland and Berkefeld filters were interposed at the outlet of the asbestos filter without avail. Finally, the difficulty was overcome by both improving the quality of the filter pad and interposing an aloxite fiber eliminator between the asbestos filter and the collecting bottle.

5. Standardization of filter pads: The compressed asbestos pads, as now prepared for our use by the Ertel Engineering Corp., are calibrated with regard to two properties: (a) The speed of filtration; and (b) degree of pyrogen retentiveness.

Speed of Filtration.—This is controlled by three factors: The filtering pressure applied; the filtering area; and the composition of the pad. The filtering pressure *is safest at 12 lbs.*, and the filtering area is limited by the size of filter practicable in hospitals. Our present filter takes pads of 12 inches, with an actual filtering area of 29.5 cm. in diameter. The pads are made of asbestos and pulp. The more asbestos used, the greater the pyrogen retentiveness, but the slower the filtration rate. The interplay of these two factors, therefore, determines not only the rate of filtration but also the efficiency of the pads to remove pyrogen. The pad now in use has a delivery volume of two liters every 2.5 minutes. A six-hour filtration would, thus, yield 288 liters, which would be more than enough to meet the daily requirements of a fairly large hospital. Hospitals using larger amounts of fluid may enlarge the filtering area of the filter by the method to be described later or install two such filters.

Pyrogen Retentiveness of the Filter Pads.—The ability of these pads to remove pyrogen was shown in a previous work[16] to be due to adsorption and not to sieving. This means that the property can be exhausted. It is, therefore, necessary so to standardize the pads that they are not only uniform but also possess such a margin of safety in pyrogen retentiveness that they remove the maximum amount of pyrogen which can occur in the distilled waters and the chemicals in use.

In order to calibrate the pads in this respect it was necessary to secure a sample of pyrogen and use it as a standard. The pyrogen used in this work was isolated from the cell-free Berkefeld filtrate of the supernatant portion of concentrated stock suspension of typhoid vaccine (1 cc. = 88 billion cells). From seven liters of this suspension, four grams of the substance was yielded. The method of isolation as well as the chemical and biologic aspects of this substance will be reported elsewhere. It is necessary, however, to state that the substance is still in the crude stage, containing six per cent nitrogen, which on further purification has been reduced to 1.6 per cent. The biologic (pyrogenic) potency of this crude sample, called "pyrogen-A," has been determined on rabbits, dogs, and men. Table II shows the effect of different doses of this preparation. If the minimum effective dose (MED) is arbi-

trarily defined as that dose, per kilo body-weight, which on intravenous injection will cause a temperature rise of 0.8° to 1.0°F., the MED in rabbits is 0.1 *gamma;* in dogs, 0.4 *gamma;* and 0.03 *gamma* in men. The reason for using this crude substance as a standard instead of the more purified substance is that the process of purification is so tedious that it may be a long time before enough of the purer substance can be available for this purpose. In any case, the biologic calibration of "pyrogen-A" makes it unnecessary to wait for the purer preparation.

TABLE II

EFFECT OF DOSES OF PYROGEN-"A" IN RABBITS, DOGS, AND MEN

Subject	Weight Kg.	Dose per Kg.	Temp. Change Deg. F.	Leukocyte Count Change x 1000
Rabbit 1	3.0	0.12	0.8	
Rabbit 2	2.5	0.12	1.0	
Rabbit 3	3.0	0.12	0.9	
Rabbit 4	3.0	2.0	3.5	
Dog 1	15.0	0.3	1.0	—6.1
Dog 2	16.0	0.3	1.1	—5.5
Dog 3	15.5	0.3	0.8	
Dog 4	16.0	2.0	3.2	—10.2
Man 1	52.0	0.04	1.2	
Man 2	64.0	0.04	0.9	
Man 3	72.0	0.04	1.1	
Man 4	70.5	2.0	5.0	

Process of Calibrating the Pads.—One milligram of "pyrogen-A" (1000 *gammas*) is weighed out on a sensitive analytic balance and dissolved in 100 cc. of pyrogen-free distilled water. Ten cubic centimeters of this solution contains 10 *gammas* of pyrogen. Amounts of from five cubic centimeters up, in multiples of five, are filtered through test pads of a convenient size mounted in convenient filters. The filtrates are then tested in both rabbits and dogs. Table III shows the protocols of some of these tests.

TABLE III

CALIBRATION OF FILTER PADS

Experimental Animal	Weight Kg.	Volume Prefiltrate Cc.	Total Dose Pyrogen in Prefiltrate *Gamma*	Temp. Change Deg. F.	Leukocyte Count Change x 1000
Rabbit 1	2.5	30	300	—.2	
Dog 1	16.0	30	300	+.1	—.2
Rabbit 2	3.0	35	350	+.2	
Dog 2	15.0	35	350	.25	— .3
Rabbit 3	3.0	40	400	+.15	
Dog 3	15.5	40	400	+.1	+ .1
Rabbit 4	2.5	45	450	+.4	
Dog 4	16.5	45	450	+.2	— .8
Rabbit 5	3.0	50	500	+.4	
Dog 5	15.5	50	500	+.8	—1.2

It will be seen from Table III that the pad removes nearly 500 *gammas* of "pyrogen-A." Since the pad is 2.5 cm. in diameter, it has a surface of 4.9 sq. cm., and, therefore, each square centimeter removes over 100 *gammas* of "pyrogen-A." The filtering surface of the four large filter pads used in the hospital filter assembly are each 29.5 cm. in diameter, making a total filtering area of 2734 sq. cm. The amount of pyrogen removable by the entire filter

assembly is approximately 273,400 *gammas*. Since about 140 *gammas* are required to cause a temperature rise of 5.6° F. in an average man of 70 kilos, this filtering area will remove about 1953 such doses.

When applied to solutions of sodium chloride or dextrose in distilled water this range of safety is more than ample as the amount of pyrogen contained in any combination of these three substances in the usual dose is tenuous and, further, the filter-pad assembly is made to deliver not more than 288 liters with each filtration. The case of sodium citrate requires special consideration. As shown in Table I, gram for gram, the contamination of pyrogen in sodium citrate is heavier. This, however, is more than offset by the fact that the dose of sodium citrate used per 500 cc. of blood is usually in the neighborhood of 1875 mg. in 75 cc. of water. This, by weight, is only about one-fifth of the dose of sodium chloride in one liter of physiologic salt solution and less than one-twenty-seventh of the dose of one liter of 5 per cent dextrose solution. Therefore, in making sodium citrate solutions, it is only necessary to use two filter pads and restrict the manufacture to not more than 300 doses.

These calculations are admittedly based on biologic effects, and assume that pyrogen from sources other than the typhoid bacilli have the same potency per unit-weight as typhoid pyrogen. This assumption has no direct experimental basis at present. The only justification for applying the figures derived from "pyrogen-A" to the pyrogenic substances found in these fluids, whose exact source is unknown, is that they have so far held true in the samples whose pyrogenic content was roughly determined by the biologic test, then filtered through asbestos pads of the required area and then retested and found negative for pyrogen. In the absence of more exact knowledge, this approximate correspondence for the present can be used as a rough working principle. This method of pretesting before filtration may be used for any intravenous material whose maximum pyrogenic content has not been as thoroughly studied as have those of dextrose, sodium chloride, and sodium citrate.

RESULTS OF 42 MONTHS' TRIAL

During this experimental period, 3867 liters of intravenous fluids prepared by the nonpyrogenic method have been administered. They may be tabulated as follows:

No. of Administrations	Amount Given Liters	Total Given Liters
1815	1	1815
652	2	1304
123	3	492
64	4	256
Totals 2654		3867 liters

In none of these was there a reaction. One patient who complained of a chilly sensation after receiving 1000 cc. did not have a temperature rise. It

is clear that if any pyrogen was present in the solutions, it was in such minute amounts as to be below the MED even in three to four liters.

Part II—Mechanical Aspects

The parts which comprise the entire filter assembly may now be enumerated and the more important items described. The filtering portion of the assembly is diagrammatically represented in Figure 1. It consists of a compressed nitrogen or air tank, A, which furnishes filtration pressure; the reservoir, B, which holds the prefiltered mixture; the filter itself, C; the aloxite fiber eliminator, D; and the collecting flask, E.

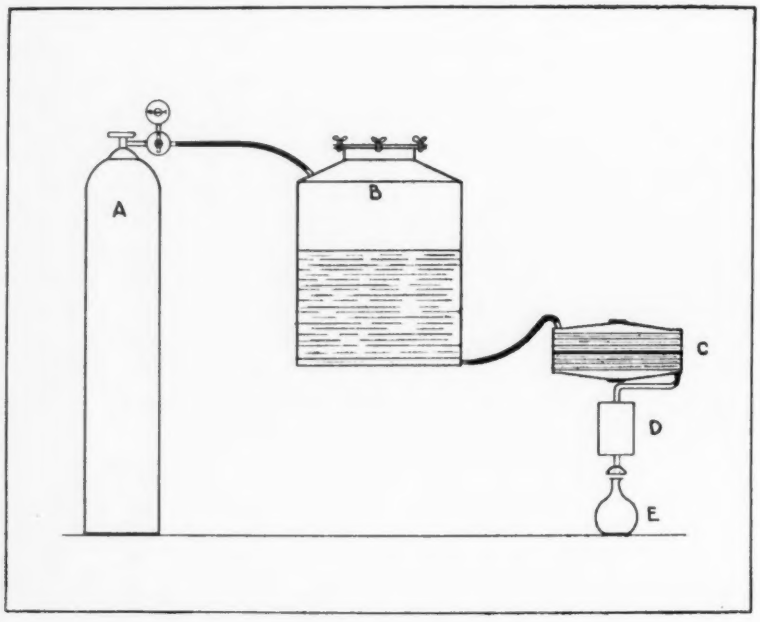


Fig. 1.

The reservoir, usually of 250 liter capacity, is made of stainless steel, and may be equipped with an electric stirrer.

Figure 2 illustrates the essential parts of the filter as well as the path taken by the solution passing through it. It may be seen that each filter pad is held between two perforated Monel metal plates or frames, the three forming a pad-frame unit. Two such units form a compartment. The fluid flows from the solution inlet, A, into a conduit system, B, which splits it into two streams. One stream filters through one pad downward and the other upward to meet in the collecting chamber, C, situated between the two pad-frames. From C the solution is conducted into the second compartment to have the process repeated, after which the conjoint stream from the second collecting chamber, D, passes through the fiber eliminator into the collecting flask. Both the latter are to be described in a subsequent section.

Since provision is made in the present filter to accommodate two additional compartments, the filter has a still wider range of usefulness than has been calculated. Any one of the three factors—capacity, speed of filtration, or retentiveness—can be increased two-fold by this doubling of the filtration area. The stream in this case would be split in four instead of two as in the filter illustrated in Figure 2.

Figure 3 is a photograph of the filter ensemble. A is the spindle with which the pads are loaded and unloaded and the filtering system rendered watertight. B is the filtering system, C is the pressure gauge, D the housing for the aloxite fiber eliminator, and E the outlet. The self-autoclav-

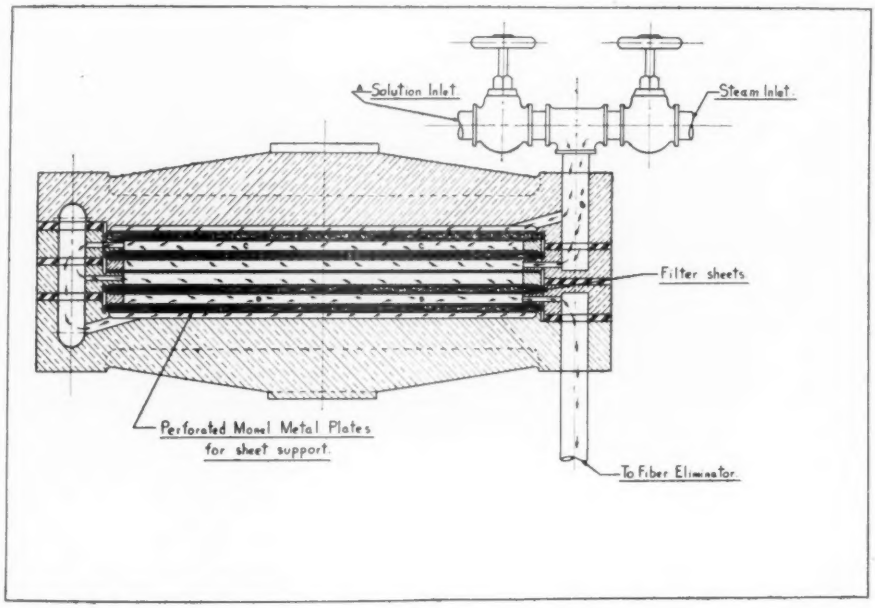


FIG. 2.

ing system, F, consists of a steam generator in which the source of steam used is pyrogen-free distilled water. It may be heated electrically, by gas, by house-steam, or by an oil burner. The steam generated is led into the interior of the filter by the flexible hose, G. The course of the steam through the filter is identical with that of the fluid which follows it and whose course has been described in the foregoing paragraph.

The aloxite fiber eliminator is a thick, finely porous filter made of carborundum. Because of its composition it is sturdy and may be heated to a temperature high enough to destroy any pyrogen which may have developed in the interstices of the stone.

The dispensing part of this system, mainly the collecting and storing flasks and accessories, is essentially the same as that described by Walter.[8, 9, 10] It is the most satisfactory one available at present, made to withstand hard

usage, and in three years of use has suffered the minimum amount of breakage. It has, however, the slight inconvenience of having too many parts.

Figure 4 illustrates the part of this system which consists of: A, pyrex flasks of specially rugged construction of one to two liters capacity, preferably graduated; B, a one-hole rubber bushing to fit the flask; C, a special stainless steel stopper with a partially grooved stem; D, a two-way vent tube for the administration of the fluids and for the admission of air into the flasks to displace the fluids.

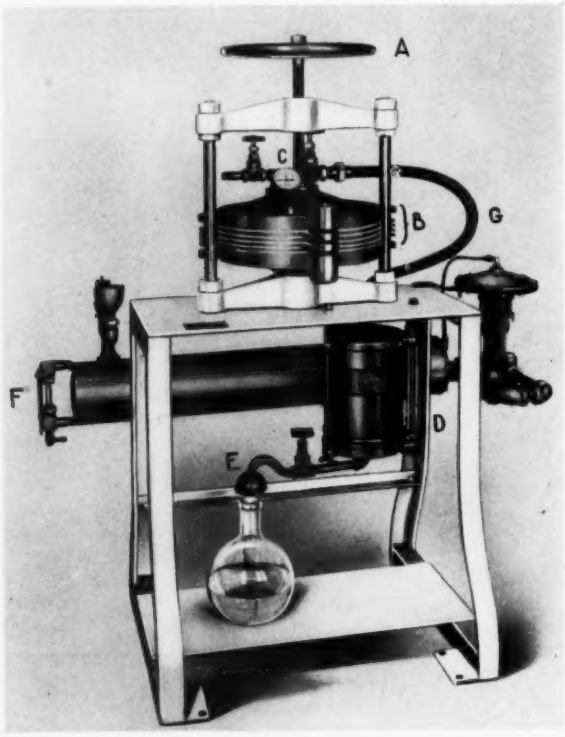


FIG. 3.

The bushing, B, is of special rubber which can stand 500 sterilizations without shrinking. It is provided with a hole in the center for the insertion of the two-way vent tube when fluid is being administered, and for the insertion of the steel stopper when the flask is not in use. The stopper is provided with a partially grooved stem which is fitted partly into the hole of the bushing while sterilization takes place. The groove allows equalization of inside pressure with outside pressure. When sterilization is completed, the stem of the stopper is pushed all the way in, as in E, the umbrella of the stopper serving as an hermetic seal for the rubber bushing.

The original Fenwal-tube is short, so that the flask end is submerged in the fluid when the flask is inverted for administration. There are two objec-

tions to this feature: First, when the infusion is being given, the air which enters the flask to displace the fluid has to be bubbled through the fluid. This, in effect, means the washing of 1000 to 2000 cc. of unsterile air in the infusion fluid. Bacteriologic cultures of ten samples of fluid subjected to this kind of air-washing gave three positive cultures. By the simple device of

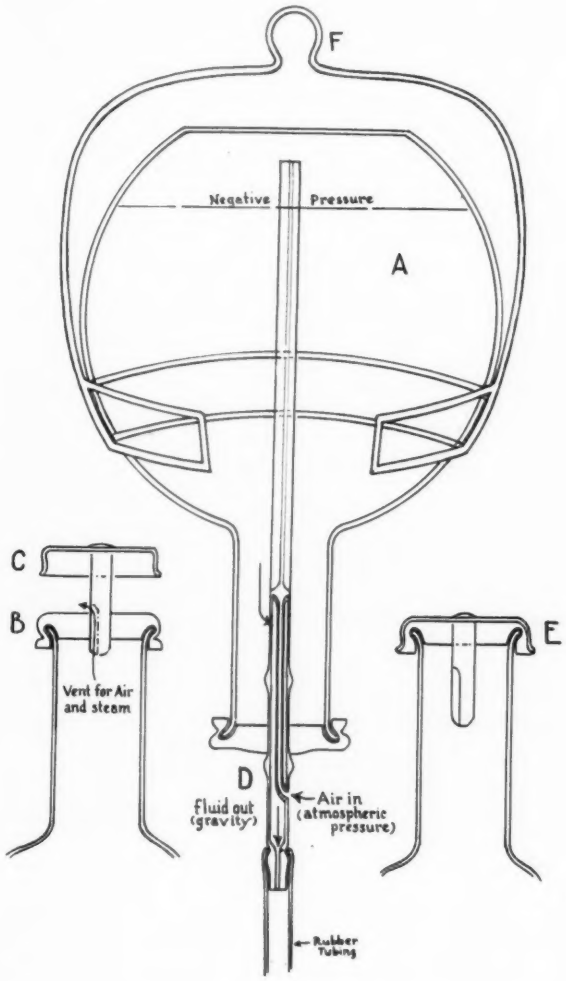


FIG. 4.

lengthening the tube so that the flask end extends above the surface of the fluid when the flask is inverted, this washing effect is avoided.

No positive cultures were found in ten fluids subjected to this system of air displacement. If it is desired to admit absolutely sterile air into the flask, the air vent may be modified in such a way as to accommodate a small sterile Berkefeld candle. This was originally used by us but later discontinued because of inconvenience.

The second objection to the short original Fenwal-vent-tube is that unless special care is taken, the fluid is as liable to flow through the air vent as through the fluid vent.

The split ring bracket, F, is simply a wire basket for suspending the flask in an inverted position when the flask is in use.

Some reference must be made to the still which delivers distilled water for making the solutions. Since the still is the heart of all other processes, much emphasis has been laid on it. While it is our opinion that the undue concentration on the still has lulled to rest suspicion of other possible sources of pyrogen, we believe that the still must be cleaned periodically so that it adds no pyrogen to the distilled water to lower the factor of safety of the filter. Moreover, it must be large enough to deliver the necessary amount of distilled water to be used for cleansing as well as to make the solutions themselves. A still that delivers 10 gallons of water per hour (40 liters), will be sufficient to meet the needs of the average hospital. The periodic cleaning also does away with incrustrations which lower the output of the still.

CARE OF THE PARTS

The Reservoir: The reservoir when new must be rinsed first with soap and water and then copiously with distilled water. Subsequent care after every use consists in rinsing it out thoroughly with distilled water and then drying it.

The Filter: When new, the conduit system of the filter is rinsed thoroughly with distilled water. Thereafter, the same rinsing process is repeated after each use.

The Aloxite Fiber Eliminator: After every use, the surface of the stone fiber eliminator is simply flushed thoroughly in order to wash away the particles and shreds which may be caught on its surface. After washing, it is placed in an electric oven at a temperature of 120° C. for one hour to dry. This temperature sterilizes the stone so that pyrogen production within the interstices of the stone is halted.

Fenwal-Flasks, etc.: The initial cleaning and subsequent care of the Fenwal-flasks, rubber bushings, steel cages, and the modified vent tube have been described by Walter and need no extended treatment here. Suffice it to say, that the flasks are washed and brushed with hot soapsuds and rinsed first in tap and then in distilled water until the walls run homogeneously clear. The same treatment should be applied to the other glassware, namely, the vent tube and the drip as well as to the steel stopper. An occasional treatment of the glassware with a cleaning mixture of 1.5 per cent solution of sodium dichromate in concentrated sulfuric acid may have to be resorted to in order to get rid of spots in the glassware difficult to eliminate otherwise. The rubber bushings when new are first scrubbed with soap and water until no further powdery substance is given off, then rinsed with

distilled water. The rubber tubings when new are likewise rinsed well with hot soapsuds and then with distilled water. Immediately after each washing, the rubber tubings, the vent tubes, the drip and the needles are wrapped and packed in a convenient container. We have used for this purpose a specially constructed copper cylinder, one foot long and 2½ inches in diameter, provided with a detachable cap as well as holes in the wall to allow penetration of the steam in the autoclave. After sterilization, the holes are closed by a special sliding device and the set stored for future use.

Preparation for Filtration: The flasks without the rubber bushings in place are sterilized in the hospital autoclave. The rubber bushings and the steel stoppers are also sterilized but separately from the flasks.

The filter with pads mounted on the frames is sterilized by its own self-autoclave for 30 minutes at 18 pounds pressure. After that it is allowed to cool to a temperature convenient for handling.

The solution is mixed in the reservoir. The reservoir is then connected, on the one hand, with the nitrogen tank and, on the other hand, with the inlet of the filter. The collecting and storing flask is placed at the outflow tube of the filter and filtration is begun by turning on the gas gradually to 12 pounds. After discarding the first four liters, the flasks are then filled one by one.

After collection, the flasks are partially stoppered with the stainless steel stopper in such a way that the groove in the stem of the stopper allows communication of the inside of the flask with the outside (Fig. 4). The flasks are then put into the autoclave and sterilized at 250° F. for 30 minutes. After sterilization, special care is taken to have the pressure in the autoclave gradually diminished so as not to cause boiling. The stem of the steel stopper may then be pushed home, in which case a partial vacuum is created as the solution cools. This partial vacuum, which helps retain the stopper in place and causes a water-hammer effect when the bottom of the flask is struck with the fist, may be used to indicate whether the stopper is leaking after storage.

After the flask is sealed, it is stored until needed.

Just before administering the fluid, the steel stopper is removed from the flask with a rocking and unscrewing motion. The flask end of the two-way vent tube is now inserted through the hole in the rubber bushing to the point where one of the two dilations on the glass tube just slips into the flask beyond the rubber bushing. The flask is inverted, placed in the ring bracket, and the infusion started.

Calculation of Costs: This method requires an initial equipment which may be divided into two parts, an irreducible item which consists of the reservoir and the filter, and an item which can be expanded according to need, namely, the flasks and their accessories. The rubber tubings and drip and needles do not enter into consideration here as they will have to be used in any case. The irreducible equipment costs about $1000. If the cost of the flasks for initial use is set at $600 and to this is added part-time labor of $50 a month, which can be expanded into full-time labor as the need arises,

the total may be set at $2200. Any hospital which expends this amount of money a year for infusion fluids will not incur a loss, even in the first year, by adopting this system. Beyond the first year the savings are substantial; the larger the amount of fluids used, the more substantial the savings, since the only items of expense will be the cost of replacements, material and labor.

But the savings are not the entire consideration. The safety from pyrogenic reactions in intravenous medication, especially in blood and plasma banks, should be the major consideration, particularly during war times. Instances are known of tens of liters of blood and plasma which have had to be discarded because the sodium citrate used was pyrogenic. This type of saving has not entered into the above calculation but may be the major item.

SUMMARY AND CONCLUSIONS

1. Methods of intravenous fluid manufacture which depend only upon the purity of distilled water used have no provision for dealing with pyrogen should this substance be present in the final mixture and, therefore, do not entirely eliminate pyrogenic reactions.

2. Chemicals have been shown to be occasionally contaminated with pyrogen.

3. The adsorptive filtration method of preparing assuredly nonpyrogenic fluids, described five years ago, has been developed into a practicable and convenient method for hospital use.

4. The compressed asbestos filter pads, around which this method is built, have been calibrated for speed of filtration and pyrogen retentiveness. The factor of safety is shown to be more than ample for the most exacting use in the manufacture of physiologic salt, dextrose, and sodium citrate solutions.

5. The uniformly nonpyrogenic results of 42 months' use of fluids manufactured according to this method are reported.

6. The mechanical aspects of the method are described.

7. The method not only eliminates pyrogenic reactions but is also more economical than the use of commercial fluids.

The authors wish to acknowledge their grateful thanks to Dr. Edward M. Berneker, Commissioner of Hospitals, to Commissioner Albert Pleydell and to Mr. A. M. Moore of the Department of Purchase, City of New York, for their encouragement and cooperation in furnishing the equipment for the completion of this work; to Dr. William F. Jacobs, Medical Superintendent of Bellevue Hospital, for the privilege of installing the experimental filter in the Bellevue Hospital, to Mr. Francis X. Dealy, of the Ertel Engineering Corp. of New York, for his cooperation in the mechanical phases of the problem, and to Dr. Wilfred Ruggiero, Messrs. Arthur Yates, Angelo Benaglia, and Milton Schrift for their constructive suggestions; to Dr. Ralph S. Muckensus, of the Bureau of Laboratories, City of New York; to Lederle Laboratories, Inc., and to E. R. Squibb & Co. we are indebted to for supplies of the stock suspension of typhoid bacilli.

REFERENCES

[1] Co Tui, McCloskey, K. L., Schrift, M., and Yates, A. L.: A New Method of Preparing Infusion Fluids. J.A.M.A., **109**, 250, 1937.

[2] *Idem:* A New Method of Preparing Nonpyrogenic Intravenous Infusion Fluids: Based

on Removal of Pyrogen by Adsorptive Filtration. ANNALS OF SURGERY, **106,** 1089-1094, December, 1937.

[3] Siebert, F. B., Fever-producing Substance Found in some Distilled Waters. Am. Jour. Physiol., **67,** 90, 1923.

[4] *Idem:* Cause of many Febrile Reactions following Intravenous Injections. Am. Jour. Physiol., **71,** 621, 1925.

[5] Hort, E. C., and Penfold, W. J.: The Dangers of Saline Injections. Brit. Med. Jour., **2,** 1589, December 16, 1911.

[6] Rademaker, L.: Cause and Elimination of Reactions after Intravenous Infusions. ANNALS OF SURGERY, **92,** 195, 1930.

[7] *Idem:* Reactions after Intravenous Infusions: Further Report on their Elimination. Surg., Gynec. and Obstet., **56,** 956, 1933.

[8] Walter, C. W.: Economical Intravenous Therapy. J.A.M.A., **104,** 1688, 1935.

[9] *Idem:* Preparation of Safe Intravenous Solutions. Surg., Gynec. and Obstet., **63,** 643, 1936.

[10] *Idem:* The Relation of Proper Preparation of Solutions for Intravenous Therapy to Febrile Reactions. ANNALS OF SURGERY, **112,** 603, 1940.

[11] Co Tui and Schrift, M. H.: A Tentative Test for Pyrogen in Infusion Fluids. Proc. Soc. Exper. Biol. and Med., **49,** 320-323, 1942.

[12] Co Tui, Schrift, M. H., McCloskey, K. L., and Yates, A. L.: Filtration Studies on Pyrogenic Inulin. Proc. Soc. Exper. Biol. and Med., **36,** 227-230, March, 1937.

[13] Smith, H. W.: Personal communication.

[14] Co Tui and Schrift, M. H.: Production of Pyrogen by some Bacteria. J. Lab. and Clin. Med., **27,** 569-575, 1942.

[15] Krno: Private communication quoted by Walter.[10]

[16] Co Tui, McCloskey, K. L., Schrift, M. H., and Yates, A. L.: Filtration Studies on Reactive Infusion Fluids. Proc. Soc. Exper. Biol. and Med., **38,** 297-300, 1936.

A SIMPLE METHOD OF PLASMA PROTEIN ESTIMATION

FREDERIC W. TAYLOR, M.D.,

AND

MARJORIE M. GIBBONS

INDIANAPOLIS, IND.

FROM THE INDIANA UNIVERSITY SCHOOL OF MEDICINE, INDIANAPOLIS, IND.

A KNOWLEDGE of the plasma protein level is desirable in all surgical cases. It is an absolute necessity in many surgical emergencies. Therefore, there is general interest in any means by which the blood plasma level may be quickly estimated.

Very frequently it is imperative to know whether or not the patient's plasma proteins have fallen below a given point. The exact plasma protein level is often unimportant. We merely want to know if the protein is about normal, less than normal, or in the dangerous range of five Gm. or less per 100 cc. of plasma. A variation of a few tenths of a Gm. makes little difference. There often may be this much variation in the individual patient.

The means here suggested by which this estimation may be carried out is not new. We have borrowed liberally from others[1, 2, 3, 4] and have merely reapplied certain previously reported principles to suit the present needs.

The method depends upon the relationship between specific gravity and protein content of a given plasma. This is applied by using tubes containing solutions of varying specific gravities. The specific gravities are adjusted so that they will equal given protein plasma levels. Therefore, when a drop of plasma is placed in the test solution, it will sink if it contains more protein than that represented in the test tube. Likewise, it will float if it contains less.

Specifically, we have made test solutions containing xylene and bromobenzene.[5] Using approximately 75 per cent of the former solution, bromobenzene was added until the correct specific gravity was obtained. This was determined by testing a portion of the mixture with a drop of potassium sulfate test solution having the desired specific gravity.

In the tests here described arbitrary specific gravities were selected. These were chosen so that they would have specific gravities corresponding with blood plasmas of 5.0, 5.8, and 6.5 Gm. per 100 cc., respectively. The specific gravity values were obtained from Weech's formula.[4]

It is simpler for the average laboratory to use serum rather than plasma in these tests. Therefore, the amount of plasma fibrinogen was substracted from the values given above. The usual value of fibrinogen is 0.27 Gm. per 100 cc.[6] Then, for example, to obtain the specific gravity of blood plasma having 6.5 Gm. per 100 cc., when using serum for the test, 0.27 Gm. were substracted from 6.5 Gm. The result is 6.23 Gm. The specific gravity corresponding to this protein content is 1.0253, when figured by Weech's formula or taken from Scudder's tables.[7]

Three such specific gravities were arbitrarily chosen and solutions were made of xylene and bromobenzene to correspond with these specific gravities. As noted, this was done by means of test solutions of potassium sulfate. These test solutions were titrated against the usual falling-drop apparatus in the laboratory. After the desired specific gravities were obtained, the potassium sulfate solutions were then used to titrate the specific gravities of the xylene-bromobenzene solutions. This latter operation was done by taking samples of the mixture in which xylene made about 75 per cent. Xylene or bromobenzene were then added cautiously, until a drop of the potassium sulfate test solution remained suspended half way in the small test tube sample. With the tubes thus carefully adjusted, they were ready for trial with the unknown blood serum.

The values selected were as follows:

Tube No.

(1) 5.0 Gm. —0.27 Gm. = 4.73 Gm. = sp. gr. 1.0209
(2) 5.8 Gm. —0.27 Gm. = 5.53 Gm. = sp. gr. 1.0231
(3) 6.5 Gm. —0.27 Gm. = 6.23 Gm. = sp. gr. 1.0253

From the foregoing it will be noted that the tests are run on serum, but the results are given roughly in Gm. of *plasma* protein per 100 cc.

Therefore, when the determination is run, a droplet of serum from the patient's clotted blood is allowed to fall into each of the three test tubes. If the serum contains less protein than that indicated on the tube, the serum droplet will float. If it contains more, it will sink.

Of course, the particular specific gravities may be altered to suit any individual requirement. The specific gravities used here seem to cover amply the needs of the usual surgical case. For instance, any blood which shows a plasma protein less than five Gm. (serum floats in this tube) indicates that the case is extremely critical, requiring emergency measures. If the serum sinks in the five-Gm. tube but floats in the 5.8-Gm. tube, the plasma protein must be between these levels. This would indicate a serious situation, but one not demanding the immediate attention cited in the first example. Likewise, we have argued that blood containing more than 6.5 Gm. per 100 cc. of plasma needs no protein therapy. This at least holds true for the surgical patient.

It is essential that the xylene-bromobenzene mixture be maintained at a known definite temperature during the tests. In the present instance they were titrated at 25° C. (77° F.). Therefore, the tests on the unknown plasma must be run at this temperature. This is necessary since the specific gravity of the xylene-bromobenzene solution changes rapidly with any change in temperature. A variation of 1° C. from 25° C. makes little difference. However, a 5° C. variation produces an error which makes the test quite useless. The temperature is easily maintained by means of a glass of water and an ordinary thermometer. The three tubes are placed into this simple water bath for one or two minutes before running the test. It is more accurate to keep them immersed while dropping the serum into them.

If plasma is used instead of serum, a substration of 0.3 Gm. (approximate) must be made from the final result.

The three tubes (Fig. 1) are used but once and then discarded. They are cheap and become inaccurate with repeated use. So that there will be no confusion in identifying the three tubes, a red dye was added to color the solutions. For this, Sudan IV was employed. The solution in the 6.5-gram tube was stained red, that in the 5.8-gram tube was stained pink, and the 5-gram tube was left colorless.

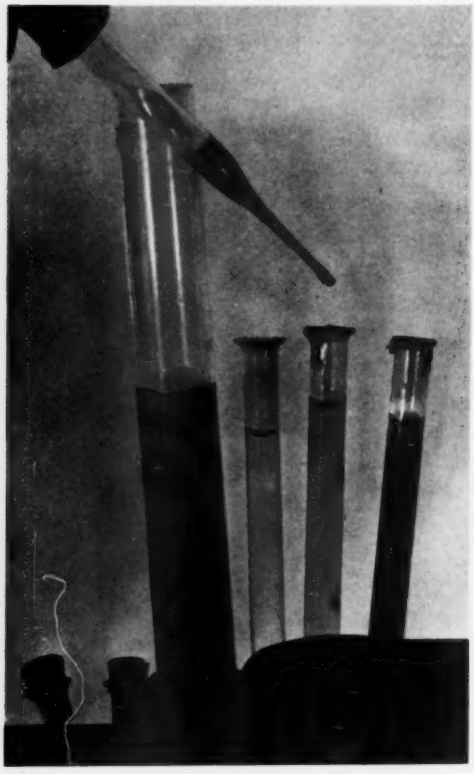


FIG. 1.—Estimation of Plasma Protein: A drop of serum from the large test tube on the left has just been placed in the center small tube. This tube has a specific gravity equal to plasma protein of 5.8 Gm. per 100 cc. Since the drop is sinking to the bottom of the tube, the serum must contain protein in excess of 5.8 Gm. per 100 cc.

The tubes were closed with cork stoppers. After closure the corked ends were dipped in a thick celluloid-acetone solution. This proved satisfactory for at least a few months. How much longer it would prevent evaporation is not known. Rubber corks were purposely avoided because of the action of xylene on rubber.

Caution: Several points of caution should be mentioned in this brief description. First of all, the specific gravities of the solutions must be extremely accurate. Carelessness in titrating the solutions may produce errors of nearly one gram in the final result.

All tests must be run at the temperature used in titrating the original xylene-bromobenzene solutions. A variation of one degree centigrade from this original temperature cannot be exceeded.

Small test tubes (2 cc.) were used for economy. This necessitated care and practice in getting the droplet of serum to fall into the solution without touching the sides of the tube. A small pipette giving small drops was an aid in this step.

Readings must be made in the first half minute or minute after the drop of serum is introduced into the solution. Occasionally, if this is not done, enough diffusion of electrolyte takes place so that a false reading is obtained.

Tubes must be opened just before the test. They are inexpensive and, therefore, can be discarded after.

Lastly, the limits of this test for plasma protein must be fully realized. It is a rough determination but possibly a life-saving one in an emergency.

SUMMARY

1. A simple test for the rapid estimation of plasma protein is outlined.

2. The test depends upon the utilization of solutions of known specific gravity. Into these are placed droplets of serum from the blood to be tested. This estimation of the specific gravity indicates roughly the amount of protein present.

3. The test is crude but accurate enough for the usual surgical patient.

4. This procedure is not suggested as a substitute for any of the methods of plasma protein determination now in use, such as the quantitative balance and volumetric pipette method, or the falling-drop method. It is suggested for use in the small hospital unit, for ships and units of the military forces where such tests are not now available. Here, it might well be a life-saving guide in cases of severe burn, trauma, or shock.

REFERENCES

[1] Barbour, H. G., and Hamilton, W. F.: The Falling-Drop Method for Determining Specific Gravity. Jour. Biol. Chem., **69**, 625, 1926.

[2] Moore, N. S., and Van Slyke, D. D.: The Relationship between Plasma Specific Gravity, Plasma Protein Content and Edema in Nephritis. Jour. Clin. Investigation, **8**, 337, April, 1930.

[3] Page, I. H., and Van Slyke, D. D.: A Simple Test for Plasma Protein Contents below the Edema-producing Level, J.A.M.A., **99**, 1344, October 15, 1932.

[4] Weech, A. A., Reeves, E. B. and Goettsch, E.: The Relationship between Specific Gravity and Protein Content in Plasma, Serum and Transudate from Dogs. Jour. Biol. Chem., **113**, 167, 1936.

[5] These chemicals were Eastman Kodak Company's No. 43 bromobenzene and T-275 M-xylene.

[6] Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice. Ed. 2, The Williams and Wilkins Co., Baltimore, 1940.

[7] Scudder, J.: Shock. J. B. Lippincott Co., Philadelphia, 1940.

OBSERVATIONS ON THE FAILURE OF HEPARIN TO INHIBIT THE CLOTTING OF BLOOD IN VITRO BY STAPHYLOCOCCI*

R. H. Rigdon, M.D.,

AND

Anne Haynes, B.S.

Memphis, Tenn.

FROM THE DEPARTMENT OF PATHOLOGY, UNIVERSITY OF TENNESSEE, MEMPHIS, TENN.

It is well known that certain strains of staphylococci will produce the clotting of citrated plasma.[1, 2, 3] These strains contain the so-called "coalase factor" which is considered to be different from the toxin that produces the hemolysis and the tissue necrosis.[4]

Much,[5] in 1908, showed that the anticoagulant hirudin did not inhibit the clotting of plasma by staphylococci. Gratia[6] also observed that both hirudin and peptone would not inhibit the clotting of plasma. Cobra venon and chlorozal-fast pink likewise do not prevent the clotting of plasma by staphylococci.[4] Walton[4] has recently demonstrated that heparin has no effect on the clotting of citrated plasma as produced by a staphylococcal Berkefeld-V filtrate.

In the present experiment heparin has been studied to determine its effect on the clotting of both whole blood and citrated plasma by staphylococci. Some of these strains contained the coagulase factor. Observations are also included on the effect of staphylococcus toxin on the clotting of blood and citrated plasma.

Materials and Methods.—Cultures of the following staphylococci were used in these studies:

Strain A — *Staphylococcus aureus* — Stock culture
B — *Staphylococcus aureus* — Otitis media
C — *Staphylococcus albus* — Otitis media
D — *Staphylococcus aureus* — Otitis media
E — *Staphylococcus albus* — Skin of arm—No lesions
F — *Staphylococcus citreus* — Skin of arms—No lesions
G — *Staphylococcus aureus* — Stock culture

All of these strains were nonhemolytic when grown on the surface of blood agar plates made with human blood. The organisms were grown in nutrient broth for 4 to 24 hours before they were added to either citrated plasma or whole blood. The amount of the inoculum was 0.1 cc.

Citrated human blood and citrated human plasma were obtained from the blood bank. It was usually the blood from syphilitic patients. The concentration of sodium citrate was 0.4 per cent. Rabbits' blood was obtained by bleeding the animals from the heart. The concentration of sodium citrate in this blood was 0.77 per cent. The rabbit plasma contained the same quantity of citrate as the blood.

* Aided by grants from John and Mary R. Markle Foundation, and the University of Tennessee.

In some of the experiments four cubic centimeters of either blood or plasma was used while in others the plasma was diluted three parts of plasma and one of saline. In each experiment the volume of each of the constituents was kept constant. Test tubes 1.5 x 12 cm. were used. They were usually kept for two hours in the water bath at 37.5° C, and then in the incubator for varying periods up to 48 hours.

The defibrinated blood was obtained from both man and rabbit. It was defibrinated with glass beads. The staphylococcus toxin was prepared by Lederle laboratories. Two one-hundredths (0.02) of a cubic centimeter of this toxin produced complete hemolysis of 2.0 cubic centimeters of a 1.0 per cent suspension of rabbits' red cells. The toxin was detoxified by heating it for five hours in a water bath at 56° C. One cubic centimeter of this heated toxin did not lyze the above concentration of rabbits' cells.

Heparin* was used in these experiments both in the presence of sodium citrate and as the only anticoagulant. When used as the latter, one part of heparin was added to three of rabbit blood. The quantity of heparin added to the citrated plasma and the citrated blood varied in the different experiments. The exact quantity is given in the protocols. One cubic centimeter of this preparation contained 10 mg. of the sodium salt of heparin.

In this experiment where suspension of staphylococci were used the organisms were grown on the surface of agar slants for 48 hours. They were carefully washed from the agar surface and again washed four times in large amounts of saline. The bacteria were then suspended in saline. The following dilutions of the saline suspension of staphylococci were used: Undiluted, 1-10 and 1-100.

Experimental Studies.—Observations on the Clotting of Citrated Blood and Plasma by the Strains of Staphylococci Used in this Experiment: The results obtained following the inoculation of the tubes of both the citrated blood and the citrated plasma with the seven strains of staphylococci are given in Table I. The rate at which the clot formed varied in the rabbit and the

TABLE I

CLOTTING OF BLOOD BY STAPHYLOCOCCI

	Rabbit Citrated Blood*					Human Citrated Blood				
	Time in which Clotting Occurred									
Strain of Staphylococcus	30 Mins.	60 Mins.	2 Hrs.	7 Hrs.	22 Hrs.	30 Mins.	60 Mins.	2 Hrs.	7 Hrs.	22 Hrs.
A.....	0†	+	+	+	0‡	0	0	+	+	+
B.....	0	0	+	+	+	0	0	0	0	?
C.....	0	0	0	0	0	0	0	0	0	0
D.....	+	+	+	+	+	0	+	+	+	+
E.....	0	0	0	0	0	0	0	0	0	0
F.....	0	0	0	0	0	0	0	0	0	0
G.....	0	+	+	+	?	0	?	+	+	+

* 3.0 cc. of the blood + 1.0 cc. of saline and 0.1 cc. of broth culture used in each tube. Water bath 37.5° C. for 3 hours, and then put into incubator at 37.5°C.

† 0 = no clot; ? = questionable clot; + = clot.

‡ Clots frequently dissolve after varying periods of time.

* This preparation of heparin is Liquaemin. It was supplied by Roche-Organon Inc., Nutley, New Jersey.

OBSERVATIONS ON THE FAILURE OF HEPARIN TO INHIBIT THE CLOTTING OF BLOOD IN VITRO BY STAPHYLOCOCCI*

R. H. RIGDON, M.D.,

AND

ANNE HAYNES, B.S.

MEMPHIS, TENN.

FROM THE DEPARTMENT OF PATHOLOGY, UNIVERSITY OF TENNESSEE, MEMPHIS, TENN.

IT IS WELL KNOWN that certain strains of staphylococci will produce the clotting of citrated plasma.[1, 2, 3] These strains contain the so-called "coalase factor" which is considered to be different from the toxin that produces the hemolysis and the tissue necrosis.[4]

Much,[5] in 1908, showed that the anticoagulant hirudin did not inhibit the clotting of plasma by staphylococci. Gratia[6] also observed that both hirudin and peptone would not inhibit the clotting of plasma. Cobra venon and chlorozal-fast pink likewise do not prevent the clotting of plasma by staphylococci.[4] Walton[4] has recently demonstrated that heparin has no effect on the clotting of citrated plasma as produced by a staphylococcal Berkefeld-V filtrate.

In the present experiment heparin has been studied to determine its effect on the clotting of both whole blood and citrated plasma by staphylococci. Some of these strains contained the coagulase factor. Observations are also included on the effect of staphylococcus toxin on the clotting of blood and citrated plasma.

Materials and Methods.—Cultures of the following staphylococci were used in these studies:

Strain A — *Staphylococcus aureus* — Stock culture
B — *Staphylococcus aureus* — Otitis media
C — *Staphylococcus albus* — Otitis media
D — *Staphylococcus aureus* — Otitis media
E — *Staphylococcus albus* — Skin of arm—No lesions
F — *Staphylococcus citreus* — Skin of arms—No lesions
G — *Staphylococcus aureus* — Stock culture

All of these strains were nonhemolytic when grown on the surface of blood agar plates made with human blood. The organisms were grown in nutrient broth for 4 to 24 hours before they were added to either citrated plasma or whole blood. The amount of the inoculum was 0.1 cc.

Citrated human blood and citrated human plasma were obtained from the blood bank. It was usually the blood from syphilitic patients. The concentration of sodium citrate was 0.4 per cent. Rabbits' blood was obtained by bleeding the animals from the heart. The concentration of sodium citrate in this blood was 0.77 per cent. The rabbit plasma contained the same quantity of citrate as the blood.

* Aided by grants from John and Mary R. Markle Foundation, and the University of Tennessee.

In some of the experiments four cubic centimeters of either blood or plasma was used while in others the plasma was diluted three parts of plasma and one of saline. In each experiment the volume of each of the constituents was kept constant. Test tubes 1.5 x 12 cm. were used. They were usually kept for two hours in the water bath at 37.5° C, and then in the incubator for varying periods up to 48 hours.

The defibrinated blood was obtained from both man and rabbit. It was defibrinated with glass beads. The staphylococcus toxin was prepared by Lederle laboratories. Two one-hundredths (0.02) of a cubic centimeter of this toxin produced complete hemolysis of 2.0 cubic centimeters of a 1.0 per cent suspension of rabbits' red cells. The toxin was detoxified by heating it for five hours in a water bath at 56° C. One cubic centimeter of this heated toxin did not lyze the above concentration of rabbits' cells.

Heparin* was used in these experiments both in the presence of sodium citrate and as the only anticoagulant. When used as the latter, one part of heparin was added to three of rabbit blood. The quantity of heparin added to the citrated plasma and the citrated blood varied in the different experiments. The exact quantity is given in the protocols. One cubic centimeter of this preparation contained 10 mg. of the sodium salt of heparin.

In this experiment where suspension of staphylococci were used the organisms were grown on the surface of agar slants for 48 hours. They were carefully washed from the agar surface and again washed four times in large amounts of saline. The bacteria were then suspended in saline. The following dilutions of the saline suspension of staphylococci were used: Undiluted, 1-10 and 1-100.

Experimental Studies.—Observations on the Clotting of Citrated Blood and Plasma by the Strains of Staphylococci Used in this Experiment: The results obtained following the inoculation of the tubes of both the citrated blood and the citrated plasma with the seven strains of staphylococci are given in Table I. The rate at which the clot formed varied in the rabbit and the

TABLE I

CLOTTING OF BLOOD BY STAPHYLOCOCCI

	Rabbit Citrated Blood*					Human Citrated Blood				
	Time in which Clotting Occurred									
Strain of Staphylococcus	30 Mins.	60 Mins.	2 Hrs.	7 Hrs.	22 Hrs.	30 Mins.	60 Mins.	2 Hrs.	7 Hrs.	22 Hrs.
A.....	0†	+	+	+	0‡	0	0	+	+	+
B.....	0	0	+	+	+	0	0	0	0	?
C.....	0	0	0	0	0	0	0	0	0	0
D.....	+	+	+	+	+	0	+	+	+	+
E.....	0	0	0	0	0	0	0	0	0	0
F.....	0	0	0	0	0	0	0	0	0	0
G.....	0	+	+	+	?	0	?	+	+	+

* 3.0 cc. of the blood + 1.0 cc. of saline and 0.1 cc. of broth culture used in each tube. Water bath 37.5° C. for 3 hours, and then put into incubator at 37.5°C.

† 0 = no clot; ? = questionable clot; + = clot.

‡ Clots frequently dissolve after varying periods of time.

* This preparation of heparin is Liquaemin. It was supplied by Roche-Organon Inc., Nutley, New Jersey.

human blood. There was also some variation in the time of clotting of the different batches of citrated plasma. The time in which clotting occurred was likewise influenced by both the age and the size of the inoculum. The clotting occurred more rapidly when 0.1 cc. of a 24-hours' broth culture was used than it did when an equal quantity of a five-hour growth was added.

The length of time in which the clot persisted following formation varied in the rabbit and the human blood. In the latter blood and also in citrated human plasma the clot frequently remained for 48 hours, at which time the tubes were discarded. The clot in the rabbits' blood began to liquefy after approximately six hours, and sometimes after 24 hours the blood had returned to a fluid state. Hemolysis began to occur in the rabbits' blood shortly after the clot developed. Extensive hemolysis was present within 24 hours. It appeared to us that the rate of liquefication of the clot in the rabbits' blood increased with the increase in the degree of hemolysis. The strains of staphylococci that failed to produce clotting also did not lyze the red cells. Essentially no hemolysis occurred in the tubes containing the human blood.

Effect of Staphylococcus Toxin on the Clotting of Citrated Plasma: In the preceding experiment it was suggested that the clots were dissolved through the effect of the toxin liberated by the staphylococci. Tubes of citrated human plasma were inoculated with the strains of staphylococci that produced clotting. When a clot was well formed one cubic centimeter of staphylococcus toxin was added. This quantity of toxin produced no macroscopic changes in the clot during 48 hours of incubation at 37.5° C.

This observation was repeated with citrated rabbit plasma, since the rabbit cells were more susceptible to the hemolytic action of the toxin. Following the appearance of a clot in the rabbit plasma 0.5 cc. of staphylococcus toxin was added to a series of tubes. An equal amount of heated toxin was added to a second series for the control. The clot produced by some strains of the staphylococci was definitely decreased in size and appeared softer in consistency after 20 hours of incubation. Staphylococcus toxin apparently had no effect on the clots in human plasma.

Observations on the Rate of Formation of the Clots in Human Plasma: It appeared from the early observations in this experiment that both the age of the broth culture and the amount of growth influenced the rate of development of these clots. To study this further one of the strains of staphylococci (G) was grown on the surface of an agar slant. The organisms were washed and suspended in saline. One group of tubes containing four cubic centimeters of citrated human plasma were inoculated with 0.1 cc. of this suspension of organisms. A second series with a 1-10 dilution, and a third with a 1-100 dilution. The tubes inoculated with the concentrated suspension of organisms were clotted after three hours of incubation. Those inoculated with the 1-10 dilution were found clotted after 18 hours, while the set of tubes inoculated with the dilute suspension were clotted only after 48 hours.

These observations would suggest, therefore, that the clotting of citrated

human plasma results from either some substance or some change in the medium produced by growing staphylococci. The strains of staphylococci that produced the clotting of citrated blood and plasma when inoculated into tubes of defibrinated blood did not produce any clotting.

Effect of Heparin on the Clotting of Citrated Blood and Plasma by Staphylococci: It has been shown in these experiments that certain strains of staphylococci when added to citrated blood and citrated plasma will produce clotting. Since heparin is an anticoagulant its effect was studied on the formation of these clots by the different strains of staphylococci. One cubic centimeter of heparin (10 mg.) was put into each of four sets of tubes. Three cubic centimeters of citrated human blood was added to one set of tubes, a corresponding amount of citrated human plasma was added to the second set and an equal quantity of citrated rabbit blood was added to the third. Each tube was inoculated with 0.1 cc. of a broth culture of the seven strains of staphylococci. Clotting occurred in the tubes with the heparin the same as in the controls in which one cubic centimeter of saline was added.

To study further this phenomenon of clotting, rabbits' blood was obtained directly from the heart and it was put immediately into a flask with heparin (1 part heparin and 3 parts blood) and four cubic centimeters of this blood was then put into each of a group of test tubes. These tubes were inoculated with a broth culture of staphylococci. Clots formed in this blood in the presence of a 25 per cent concentration of heparin as readily as they did in citrated rabbit blood in which saline was substituted for the heparin. It was evident, therefore, from these observations that the failure of heparin to inhibit the clotting of both citrated plasma and citrated blood in the presence of certain strains of staphylococci was not the result of the neutralization of the heparin by the sodium citrate.

Discussion.—Certain strains of staphylococci when inoculated into citrated human plasma and citrated rabbit blood produced a clot. This clotting of citrated blood was similar to the clotting of citrated plasma by the "so-called" coagulase producing strains of staphylococci. Heparin when added to both citrated blood and citrated plasma in a 25 per cent concentration had no inhibitory effect on the formation of the clot. Other anticoagulants such as, hirudin, cobra venon and chlorazol-pink likewise have no inhibitory action on the formation of clots by staphylococci in citrated plasma.[4, 5, 6]

It was necessary for fibrinogen to be present in the blood for clotting to occur when the medium was inoculated with staphylococci. This was experimentally shown by the failure of clotting to occur in defibrinated blood from both man and rabbit. Some change occurred in the blood in the presence of growing staphylococci to produce this clot. The rapidity in which the clot formed was influenced by the size of the inoculum. Rabbits' citrated blood and plasma were clotted more rapidly by the staphylococci used in this study than the citrated human blood and the human plasma. Citrated human plasma from different individuals also clot apparently at different rates by the same strains of staphylococci.

The substance responsible for this clotting of blood and plasma was not present in the preparation of staphylococcus toxin used in this experiment. Walton[4] observed that a Berkefeld-V filtrate of 2 to 14-day cultures was active in the clotting of citrated plasma. He tested the filtrate of ten strains of staphylococci, eight of which clotted before filtration, and found that none of them were active in the clotting of plasma. These observations suggest, therefore, that the filtrates from only certain strains of staphylococci can produce the clotting of citrated plasma. The dissolution of the clot in rabbits' blood, accompanied by hemolysis, suggested that this process of clotting and that of hemolysis were produced by different substances. Walton has stated that "the clotting principle is distinct from the toxin responsible for local inflammation and fixation."

The dissolution of the clot in rabbits' blood and plasma apparently occurred as the result of the effect of the toxin liberated by the growing staphylococci and also by the toxin present in the filtrate used in this study. The failure of any dissolution to occur in the clots in the human blood was interesting in view of the fact that human red cells were not lyzed by the strains of staphylococci used in this study. Red blood cells from man are usually more resistant to the hemolytic action of staphylococci than rabbit red cells.[7]

It may be significant that heparin has no effect on the clotting of blood as produced by different strains of staphylococci. Heparin is now being used in conjunction with chemotherapeutic agents in the treatments of certain types of staphylococcal infections.[8] If heparin acts similarly *in vivo* and *in vitro* its effect on thrombophlebitis may be expected to differ with the different types of staphylococci.

SUMMARY

The "coagulase" strains of staphylococci produce clotting of citrated blood from both man and rabbit.

The clot that is formed in rabbits' citrated blood by these strains of staphylococci gradually dissolve. This dissolution apparently results from the action of the toxin liberated by the growing bacteria on the clot.

Heparin in a 25 per cent concentration does not inhibit the clotting of blood as produced by the "coagulase" strains of staphylococci.

REFERENCES

[1] Loeb, L.: The Influence of Certain Bacteria on the Coagulation of Blood. Jour. Med. Research, **10**, 407, 1903.

[2] Chapman, George H., Conrad, Berens, and Merrit, H. Stiles: The Coagulation of Plasma by Staphylococci. Jour. Bact., **41**, 431, 1941.

[3] Dienst, R. B.: A Study of Recently Isolated Strains of Staphylococci and their Ability to Coagulate Human Plasma. Jour. of Lab. and Clin. Med., **27**, 663, 1942.

[4] Walton, H. D.: The Clotting of Plasma Through Staphylococci and their Products. Jour. Hygiene, **35**, 549, 1935.

[5] Much, H.: Über eine Vorstufe des fibrinfermentes in Kulturen von staphylokokkus aureaus. Biochem. Ztschr Bd. **14**, 143, 1908.

[6] Gratia, A.: Action coagulante du staphylocoque sur le plasma hirudine. Compt. rend. Soc. de biol., **82**, 1393, 1919.

[7] Rigdon, R. H.: Hemolysis Produced by Staphylococcus Colonies and Toxin on Agar Media Containing Various Animal Bloods. Jour. Lab. and Clin. Med., **24**, 1264, 1939.

[8] Schall, LeRoy A.: Treatment of Septic Thrombophlebitis of the Cavernous Sinus. J. A. M. A., **117**, 581, 1941.

DRAINAGE AND WOUND CLOSURE TECHNIQUE IN APPENDICITIS OPERATIONS

HUGH AUCHINCLOSS, M.D.

NEW YORK, N. Y.

DEATHS FROM APPENDICITIS throughout the United States continue to be appallingly high. Deaths from appendicitis in many of the large hospitals in the United States are extraordinarily low. This indicates that technique and experience are important. An appendicitis operation may be one of the easiest operations in surgery, or one of the most difficult. Entirely too many operations for appendicitis are being performed in the United States by doctors who lack this skill in technique and experience.

Problems of wound closure depend a good deal on wound-making and the necessity for drainage. Incisions for appendicitis should be made as close to where the appendix is located as possible. The minimal amount of damage should be done to the abdominal wall. They should be made so that drainage, when necessary, will be effectual and as free from postoperative complications incidental to introduction of foreign body material into the peritoneal cavity as possible.

Variations in the situation of the appendix are well known. Recognition of where the appendix is situated (Fig. 1) can be gained before operation, to a considerable degree of accuracy, by a careful study through (1) percussion tenderness, carried out with an even degree of gentle pressure; (2) deeper, graded, finger-tip pressure; (3) rectal examination; and (4) the study of a plain roentgenogram of the abdomen, which in a large number of cases, will show a characteristic gas shadow of the cecum and ascending colon.

The best incisions for acute appendicitis operations are illustrated in Figure 2. The McBurney incision is unquestionably the best incision unless there be some convincing reason otherwise. It can be made slightly high or slightly low. Where more exposure is needed, especially toward the midline, the Weir extension, through the linea semilunaris into the rectus sheath, with retraction of the rectus toward the midline, often makes a difficult operation relatively easy. In certain cases of doubt as to diagnosis, especially if a resection of the right colon may be needed, a transverse incision, (Fig. 2C) just above the iliac crest to just below the umbilicus is a good one. In certain cases where the diagnosis may be questionable, or there is indication of a large pelvic abscess, with extensions to the right and left iliac fossae, a midline incision (Fig. 2D) may be indicated. The so-called right rectus incision should be mentioned only to be condemned, not only from the standpoint that drainage through such an incision creates a drainage tract with coils of small intestine on all sides, but also because the drains are liable to injure the superior mesenteric vessel to the small intestine and the right colon. The posterior sheath and peritoneum of a right rectus

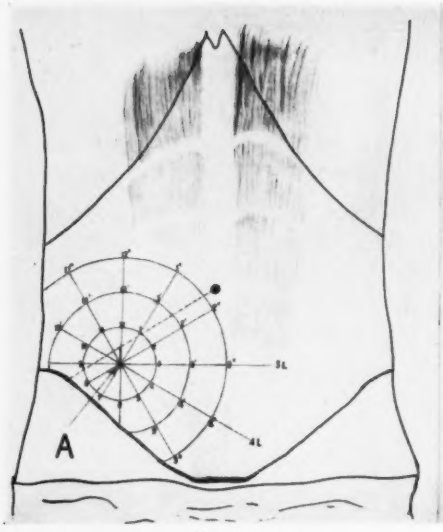


FIG. 1.

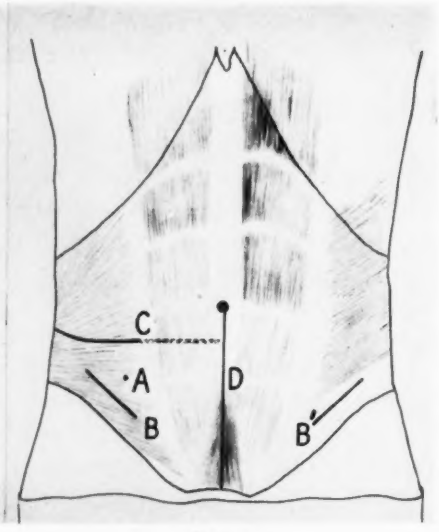


FIG. 2.

FIG. 1.—Use of the "clock dial" for convenience of description in appendicitis. McBurney's point, one-half inch below the junction of the outer thirds of a line from the umbilicus to the anterior superior spine, is used as a center. The numbers, e.g., 12, 12', 12", at the intersection of the radiating lines with the circles are used for localization descriptive purposes. The outer circle reaches to the midline below the umbilicus. Points to the left of the midline have a capital L added.

In appendicitis records, especially for anyone making an intensive study of the subject, the "o'clock" method for precision in charting symptoms, physical signs, operative technic, and pathologic findings, has a number of rather obvious advantages.

INCISIONS

FIG. 2.—A. McBurney's point: One-half inch below the junction of the outer and middle thirds of a line from the anterior superior spine to the umbilicus.

B. B'. McBurney's incision: On the left side this incision is shown to be over the aponeurotic portion of the external oblique. On the right side, it is shown in relation to the internal oblique. At this level the internal oblique fibers are transverse. The transversalis muscle is transverse throughout, so that at this point direction of the two muscles is the same.

Common faults in making this incision are (1) the skin incision is made unnecessarily long. Skin stretches more than one anticipates after the deep fascia is cut; and (2) it is made too near the midline, often in the linea semilunaris and the rectus sheath unintentionally. If drainage be required, it is well, in the great preponderance of cases, to have the drains well away from the midline and small intestinal coils.

When convincing evidence of abnormal situation of the appendix exists, the incision can be made a little high or a little low, or a Weir extension into the rectus sheath made according to obvious demands.

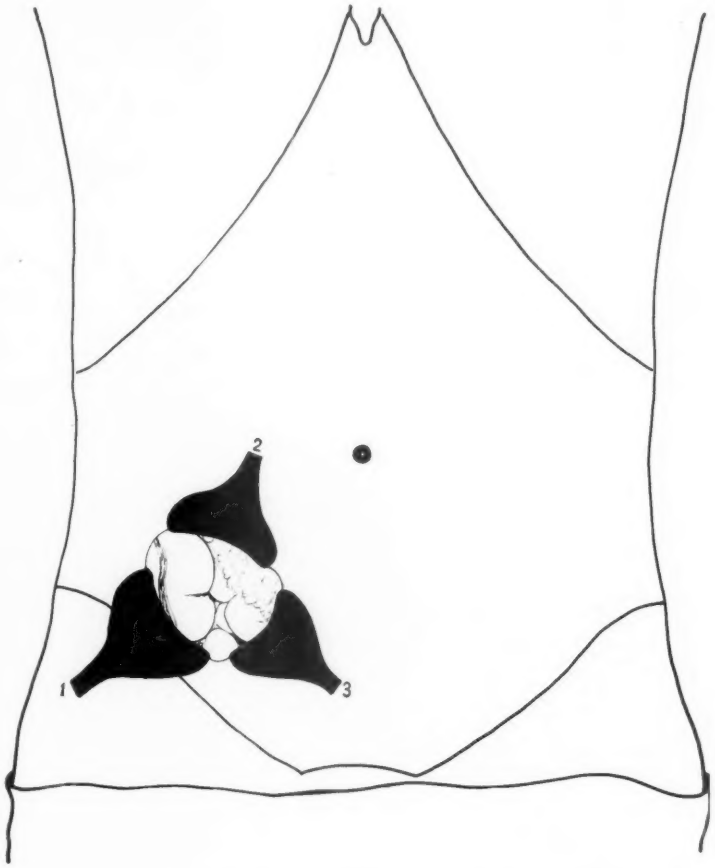
C. Transverse incision: Suited for cases where the cecum has not descended and the appendix is high, or when the appendix is retro- or laterocolic, or where a lesion that may require resection of the right colon is difficult to distinguish from appendicitis.

D. Midline incision: When accurate diagnosis is impossible, the signs preeminently pelvic, or pelvic organ disease not unlikely, this incision gives the best exposure with the least disturbance to the highly specialized neuromuscular structures of the abdominal wall. It should be borne in mind, however, that where the cecum is in its normal iliac fossa position, and an inflamed appendix tip in the pelvis, the McBurney incision, with or without the Weir extension, is the incision of choice.

One should differentiate between two associated factors in determining the incision best adapted for appendicectomy: First, the site of the cecum; and second, the site of the appendix itself. A preoperative roentgenogram may be of great assistance in determining the cecal site, but the clinical signs may be of greater value in determining the site of the appendix. A cecum situated normally in the right iliac fossa may be associated with an appendix whose inflamed tip may be under the liver or deep in the pelvis. Easy access to the cecum helps appendix removal. Easy access to the appendix—especially its tip—also aids removal.

Thus, a preoperative roentgenogram, that almost always will show gas shadows indicating the site of the ascending colon and cecum, coupled with the findings on clinical examination, may considerably help determine the best site for an incision that will provide best exposure for removal and least harmful site for accurate and adequate drainage. Such reasoning and forethought may save life. Indeed, it is its lack that, more often than we think, may be the one determining factor that makes it impossible for a case to get well.

incision may be very difficult to close, especially if there be distention. Omental and intestinal adhesions to this imperfect closure occur, especially when this incision is made at the level of the umbilicus and below. The right rectus incision is responsible for a large number of postoperative intestinal



"THREE-POINT" RETRACTION FOR SIGHT

FIG. 3.—"Seeing the situation" should be the surgeon's first thought when the peritoneum is opened. A good exposure aids, more than anything else, in finding the appendix quickly and removing it with minimal harm. Two retractors may give a good view, but three are better. Delicacy, precision and using the eyes and head to guide the hand is priceless. The good surgeon is always a gentle man.

obstructions. It would be wise to do away with the right rectus incision in the middle and lower abdomen entirely.

After opening the peritoneum through a McBurney incision, the first thing an operator should remember is to study his problem by sight. The practice of immediately putting one's finger through a small opening and searching for an inflamed appendix with the finger-tip and then making an effort to pull it out, regardless of its pathologic condition, is bad. Three-

point retraction (Fig. 3) provides an excellent method for seeing, before doing. The cecum, or ascending colon, should first be looked for. They are recognized, of course, by means of longitudinal striae. Once found, these striae lead to the appendix. In making this search retraction should be outwards, because the cecum and ascending colon, lie usually along the lateral abdominal wall, posteriorly. The appendix stump should be inverted without ligation. It is well known that other methods are advocated by many, but they are not as good surgery.

It often takes much skill and experience to remove a badly infected appendix, especially with abscess formation. Much has been said recently about draining the abscess and leaving the appendix in. There are occasional cases where this is advisable. This is where skill and experience count. The proper thing to do is always to remove the appendix unless there is convincing evidence that it is wiser to leave it in. Where there is skill and experience, the appendix can practically always be removed, and if done carefully and accurately, it is much the better thing to do.

The reasons for drainage are not always too obvious, and the trite remark "when in doubt, drain," has a certain quality of value. In many cases, a careful history, as to the *length of time* serious infection, with likely spread into the mesentery and surrounding parts, enough to cause necrosis of tissues in the neighborhood, that cannot be removed, is of value. The writer recalls assisting the late Dr. Joseph Blake, over 30 years ago. He removed a gangrenous appendix, and sucked out considerable real pus from the pelvis and peritoneal cavity, yet closed the wound. At that time the writer was amazed. The boy got well, without complications. The point was that the boy had felt apparently perfectly well only four hours previously. He had hardly had time to develop secondary necrosis in the tissues outside of the appendix. The necrotic tissue had been removed by the removal of the appendix, and in spite of the fact that the pus was almost creamy, the tissues, left behind, retained their nutrition adequately to care for the brief contamination they had been submitted to. Contamination precedes infection. Infection is dangerous. Infection that has had long enough time to make dead tissue is still more dangerous. Factors that contribute to infection, that leads to devitalization of tissues left behind, provide reasons for drainage. Long operations, much handling, many ligatures, colon-content contamination, foreign bodies, insecure inversion, exposure of retroperitoneal tissues, that are far less able to care for infection than the peritoneum itself, evidences of mesenteric thrombosis with its associated devitalizing effects and true abscess formation, all of these, are reasons for drainage.

Useful principles and methods of drainage are indicated in Figures 4, 5, 6 and 7. Where there is little evidence of necessity for draining the peritoneal cavity, but where the infection has been considered a serious one, especially in stout people, and where resistance may have been impaired, drainage of the wall either to the peritoneum or to the aponeurosis is often safer than closure. There are times when a loosely packed cigarette

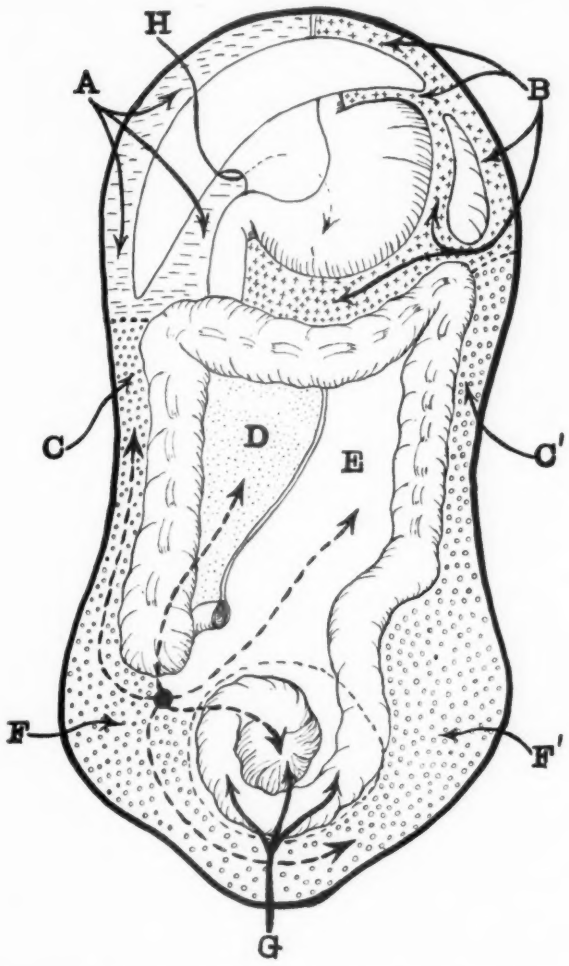


FIG. 4.—Schematic drawing (after Testut: Traite' d'Anatomie Humaine) showing the principal pouches of the peritoneal cavity. The dotted lines indicate the routes along which infection from an inflamed appendix travels, largely according to the position of the appendix with reference to the cecum. Quite apart from these intraperitoneal extensions, are the extraperitoneal, when the appendix lies behind the cecum, ascending colon or ileocecal junctions. Such retroperitonal infections toward kidney, pancreas, and along the great vascular routes are equally important and present serious problems.

One must understand these routes of infection dissemination in order to operate and drain intelligently, as well as appreciate the likely localization of, and approach to, secondary abscesses.

A. Hepatic fossa: This includes the right subphrenic, and subhepatic abscesses.

B. Gastric fossa: This is in front of the stomach, and includes the perisplenic and left subphrenic spaces.

C and C'. Right and left parietocolic, or laterocolic spaces: These communicate with the hepatic and gastric spaces above and the right and left iliac fossae below.

D. Right, mesenterocolic space: Infections from appendices in front of the ileocecal region—a relatively rare position—extend to the right of the mesentery, in contrast to

E. Left, mesenterocolic space. Infections to this region are more common because of the relative frequency of appendices pointing toward the pelvis and in this direction (Fig. 5).

F. and F'. Right and left iliac fossae: Most appendix abscesses, of course, are in the right iliac fossa. Extensions to the left iliac fossa from the pelvis in front of the rectum along the left leaf of the mesosigmoid are not uncommon.

G. The pelvis: The rectum forms more of a dividing line in the pelvis than is generally appreciated. Abscesses on the right of the rectum may extend up and into the left mesenterocolic space E. When the abscess has extended to in front of the rectum and toward its left side, the extension is to the left iliac fossa F'.

H. The foramen of Winslow leading from the hepatic space into the lesser sac: Abscesses of the lesser sac from appendicitis are practically unknown.

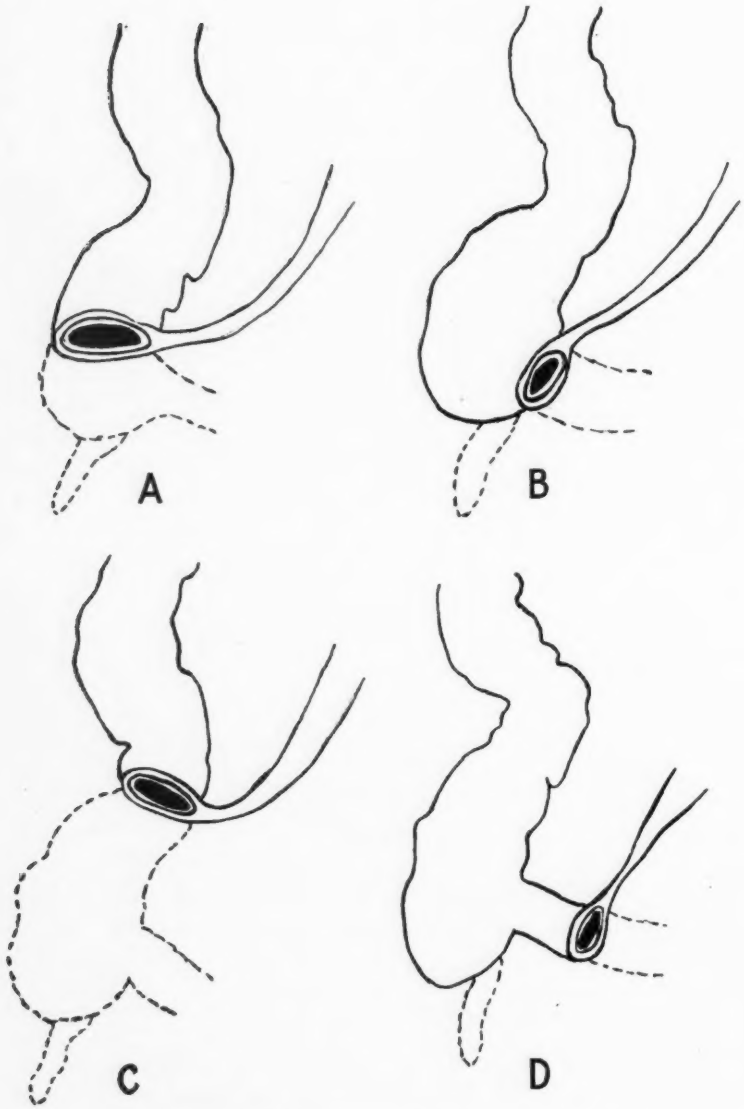


FIG. 5.—Variations in the attachments of the lower pole of the mesentery. (d'apres Turnesco,-Traite' d'Anatomie Humaine: Testut).

A. Cecocolic junction: This is the classical, normal arrangement.
B. At ileocecal valve.
C. Ascending colon.
D. Terminal ileum.

These variations, especially A, B and C, show how readily dissemination of infection to the pelvis and to the left mesenterocolic space (Fig. 4 E) can occur in the majority of cases. Of course, in the laterocolic, retrocolic and antecolic appendices, this rule does not obtain.

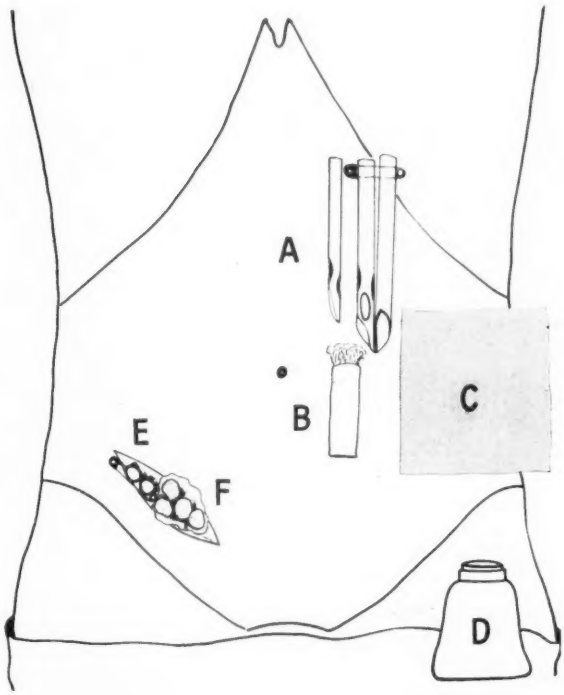


FIG. 6.—Appendicitis with abscess drainage through a McBurney incision: How to drain and leave a "wide open" wound "closed" without suture:

A. Fairly large, rubber tubes with soft walls. These are fenestrated and beveled, and used single or double. One limb of the double tube is cut a little longer than the fenestrated limb, and left without holes. This can be used subsequently for irrigation purposes. A loosely packed piece of gauze tape can be used for a few hours in the fenestrated tube to prevent a blood clot from filling the tube. The inner ends of these tubes should be accurately placed, not necessarily to where purulent exudate is found, but where the tissues have been killed by the infection and slough has already occurred, or is expected, to occur. Early removal of such tubes is not to be expected. They must be left in long enough to maintain a tract until sequestration and separation of the dead sloughs from the living tissues is complete. Gently loosening them and cutting off their protruding ends, bit-by-bit, making sure that their inner ends still reach the bottom of the sinus without causing undue pressure—letting them "shorten themselves"—is an art requiring judgment and experience.

B. Cigarette drain: Gauze should not protrude from the inner end of this drain. It is not as good a drain as a tube. It is a "plug" and, as such, is useful in keeping wounds open with the minimal risk of causing pressure necrosis. Where infection necrosis is minimal, when the length of time has been so brief as to make it seem unlikely that, though the appendix may be necrotic, the tissues left behind, after its removal, are not seriously devitalized, a cigarette drain may serve the purpose of maintaining the wound open for about 48 hours, with less likelihood of causing pressure necrosis than a tube. There are probably a certain number of cases where the proper use of tubes has been the determining factor in saving the patient's life and the use of cigarette drains inadequate. On the other hand, it should be clearly understood that the improper use of tubes can do just the opposite. It is the way in which they are used, nice precision, and experience that count.

C. A sheet of thin China silk. This, fenestrated or not, is far preferable to gauze, when used against the tissues, to keep the wound open yet prevent evisceration, as the inner lining of a tampon. One or more short cigarette drains, used inside silk, make an excellent tampon and are easily removed or replaced without causing pain or hemorrhage.

D. A salt shaker is employed for convenience in dusting the crystals of sulfanilamide, sulfathiazole, or other medication, on or in the silk tampon, or in the wound.

E. The tubes in place with a safety pin beneath which dressings can be placed to prevent their being pushed in further than intended.

F. Four cigarette drains, giving bulk to the tampon, inserted just through the peritoneum to maintain the unsutured wound wide open and prevent evisceration.

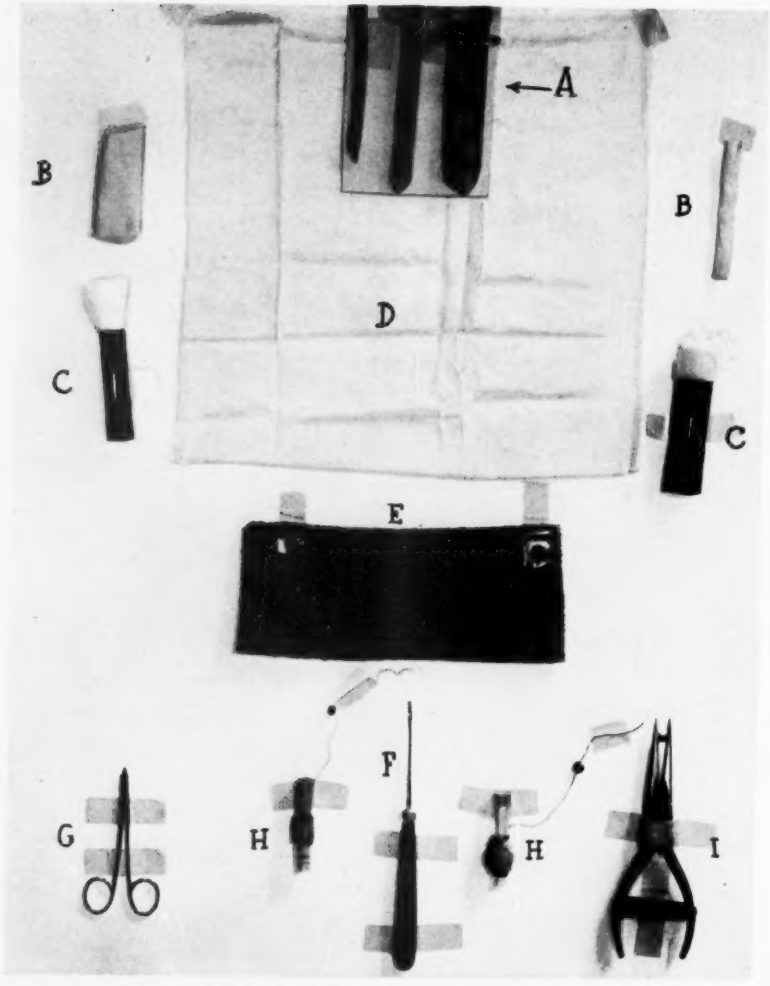


FIG. 7.—Drains and materials used for "staple-suture" wound closure:

A. Small, large, soft, single, and double rubber tubes.

B. Silk containers filled with gauze. Crystals of sulfanilamide, or sulfathiazole, etc., can be smeared on the outside or put inside these containers and used as replacement drains in the sinuses after removal of the tube drains.

C. Cigarette drains. Gauze does not protrude from their inner ends.

D. A sheet of China silk to be used for tamponade.

E. A sheet of sponge rubber. A roll of this wrapped in boric ointment gauze provides a good buffer on which to seat the shot fasteners on the skin surface bearing of the "staple" suture described under Fig. 8.

F. Reverdin needle. This, or larger skin needles, are useful in placing "staple" sutures.

G. Small cuticle clipper useful in making the split-shot. Even better yet are pruning shears, one blade of which is sharp, the other flat.

H. Strong silk sutures with split-shot fasteners.

I. A split-shot held by a pair of pliers. An elastic band about the handles holds the shot in place before crushing.

drain (Figs. 6B and 7C) a short distance into the peritoneum, to be removed within 24 or 48 hours, may be advisable. In considering the serious infections, however, of the peritoneal cavity, soft rubber tubes (Figs. 6A

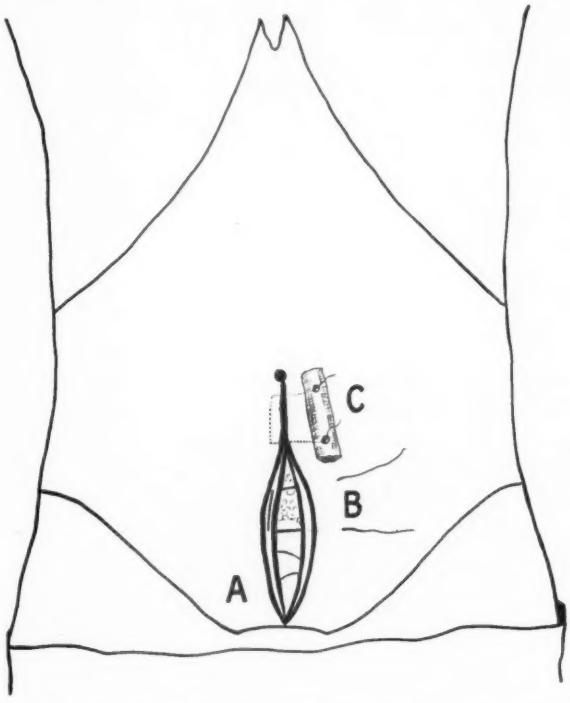


FIG. 8.—The "bolt-", or "staple"-type of suture for wound closure:

A. The lower part of a midline abdominal wound to be left open for drainage.

B. The suture. Strong silk passes through the skin on the left side through the anterior sheath and peritoneum, and often through the inner portion of the rectus, then through the peritoneum and anterior sheath on the right. The tough fibers of the anterior sheath (linea alba) if the incision has been carried slightly to the left of the midline, is used to bear the burden of the strain. This, after all, is the one structure in this wound capable of doing so. Two or three centimeters away, the suture is passed in reverse direction. This brings the opposing bearing on the skin surface.

C. This shows the wound closed and a method for handling the skin surface bearing. A roll is made of sponge rubber wrapped in boric ointment gauze. The sutures are passed through this and fastened by split-shot. Often the skin falls together in correct apposition so that sutures are unnecessary. In infected cases the subcutaneous tissues can be left open without weakening the closure.

From the standpoint of time consumed, simplicity, minimal strangulation of tissue, number of sutures, strength of closure, avoidance of wound infection, and the amount of "foreign body" material left in the wound, this method is best, in my experience.

and 7A) are the best. They provide a space in the depths of the peritoneal cavity comparable to the surface of the body into which exudate can immediately accumulate under minimal tension. For many years now, it has been recognized that closure of the wound about such drainage, where the infection has been a serious one and there has been much necrosis, is inadvisable. However, if the wound be not closed, evisceration may take place alongside

the tubes. A good way to prevent this is the use of a China silk tampon (Figs. 6C, F and 7D, C) into which have been inserted cigarette drains packed with somewhat more than the usual amount of gauze that is not allowed to protrude from their inner ends. These give bulk and can be easily and painlessly removed in a few days because they do not stick. The silk tampon is subsequently removed with minimal disturbance to the tissues it comes in contact with; far less than if gauze were used.

The use of sulfanilamide, or sulfathiazole, and the newer similar drugs (Fig. 6D) locally, has been pretty well proven of real value from the standpoint of their bacteriostatic effects. It can be dusted as a fine powder on the silk tampon (Fig. 6F) described above, after the silk has been wet with salt solution, or directly into an abscess cavity, or over the tissues of the wound in the abdominal wall. In similar fashion these drugs can be used on long, finger-like sacs made of silk and filled with gauze tape (Fig. 7B) for subsequent dressings, especially after the tubes have been removed. Wounds treated this way are certainly freer of foul-smelling, "colon" pus, and heal more rapidly and with less necrosis than heretofore.

The use of a tampon to prevent evisceration when it is desirable to have a small wound wide open, is usually adequate, even though distention occurs. This is not so in a larger wound, such as in some midline wounds. Figures 8B, C and 7E, F, G, H, I, indicate the principles that make the "bolt-" or "staple-type" of suture reliable. This gives the strongest closure with the fewest sutures and least necrosis. The wound can be left wide open for drainage, or not. If used correctly, even the skin edges may be so nicely apposed as to need no sutures.

IMMEDIATE SKIN GRAFTING IN THE TREATMENT OF BURNS

A PRELIMINARY REPORT

FORREST YOUNG, M.D.

ROCHESTER, N. Y.

FROM THE DEPARTMENT OF SURGERY, UNIVERSITY OF ROCHESTER, SCHOOL OF MEDICINE AND DENTISTRY, ROCHESTER, N. Y.

IN February, 1941, Mason[1] made a plea that burns be regarded as surgical wounds; and thought that results might be improved if the same principles used in treatment of other traumatic wounds be followed. These principles of cleansing, débridement, hemostasis, closure, and rest, are well known and generally accepted. Moreover, it is known that they hold only if wounds are cared for within a few hours following injury; preferably within six hours, certainly not longer than 12 hours. It is probable that this simple view of burns has not previously obtained because of the wide extent of the injury and the poorly understood cause of the shock found in severe burns.

Without going into the various theories of shock production, it now seems probable that shock in burns is, for the most part, due to fluid loss from the burned surface, and into the tissues about and beneath the burn; this loss of fluid accounting for the decrease in circulating fluid and protein and relative increase in blood cells. With this better understanding of burn shock, it is now possible to more adequately control it by plasma administration guided by frequent hematocrit, and plasma protein determinations. In fact, if the burn is seen soon, it is possible, within certain limits, to forestall shock by replacing plasma as it is lost. It, therefore, becomes possible, in even extensive burns, to save patients whose injuries would formerly have caused death from shock within a few hours of the injury.

The extent of the wound and its treatment then becomes increasingly important, for we are now able to save patients with wounds of much greater surface-area. But with the partial solution of this problem another has appeared. These patients may be successfully carried through the initial shock period only to succumb as long as one to three months later. This late death is probably not entirely due to infection, although this is the generally accepted view. It may be that there is a loss from these extensive open wounds of necessary body constituents not as yet understood.

There are various facts which support this view. Extreme emaciation develops in these patients with large granulating surfaces, and they may die in this stage even though infection seems minimal. This is illustrated by the following brief case report:

Case 1.—S.M.H., No. 167072; G. S., white, male, age 35, received deep third-degree burns of the entire trunk and most of the upper extremities, and first- and second-degree burns of the face and hands. The area involved, as estimated by Berkow's charts, was 55 per cent of the body surface. The burn occurred one to four hours before admission.

The exact time was not known as the man had been found in an alcoholic stupor on a burning davenport. He was in deep shock—B.P. 60/0, Hb. 23.5 Gm.; hematocrit 63.2. Within the next 64 hours he received 5800 cc. of plasma and 7500 cc. of saline intravenously before his blood pressure stabilized at 120/60. The burned areas were tanned but became grossly infected. On the ninth day he was started on tub baths in an attempt to remove necrotic tissue and control infection. The temperature chart showed continual improvement and the granulations became quite clean. In spite of this he became emaciated and very weak. He steadily lost weight and strength in spite of the fact that the lesions appeared cleaner. Many areas were perfectly healthy and pinch-grafting was started. During the fourth week he became irrational and delirious, with high fever, and a blood culture showed *B. aerogenes,* 260 colonies per cc.. He expired on the 30th day.

We have observed that the fever curve of a patient comes down in step-like plateaus as a large granulating surface is covered at intervals.

Case 2.—S.M.H., No. 37348; H. S., white, male, age 11, received a total third-degree burn of the left lower extremity from hip to ankle, a second-degree burn of the medial surface of the right lower leg from knee to ankle, first- and second-degree burns of the hands, and first-degree burns of the face. The total body area involved was estimated as 30 per cent. He was seen one hour after injury. He was not in shock—B.P. 110/68, hematocrit 50.9, Hb. 16.5 Gm. The serious nature of the burn was not recognized by the house officer who saw him, and he was started on saline compresses, without intravenous therapy. Moderate shock developed and was controlled by plasma administration. He developed severe infection in the necrotic tissue. This was gradually controlled by saline compresses and saline tubs. It was 40 days before the necrotic tissue had separated and granulations enough appeared so that skin grafting could be considered. He was running a septic type of temperature, averaging about 39°C. (102.2°F.). About one-quarter of the left lower extremity was covered with split-thickness grafts. The temperature averaged 38.5°C. (101°F.) after this. Ten days later another quarter of the leg was covered, and immediately the temperature plateau fell to 38°C. (100.4°F.). Thirteen days after this, most of the remaining open half of the extremity was grafted, and the average temperature curve dramatically and promptly fell to 37.5°C. (about 100°F.).

This boy during his illness became extremely emaciated but survived and over a period of two to three months, gradually returned to normal.

It is common experience that there is improvement of the general condition of the patient if a large granulating surface is covered with homografts—even though these grafts, as is well known, persist only from two to six weeks.

It would seem then that if Mason's view, that a burn is a traumatic wound, is accepted, that treatment should be directed toward an attempt to secure primary healing. This, if possible, would eliminate these delayed deaths caused by the open wound. We believe that the various methods of local treatment of burns now in use do not meet this requirement.

Tanning.—There are many agents used to produce an eschar over the burned surface. The one most commonly used is tannic acid. The objective of these agents has been stated as: (1) To prevent fluid loss by sealing the surface; (2) to prevent the absorption of decomposition products of the dead tissue; (3) to provide a comfortable dressing which need not be changed; and (4) to prevent infection. From the standpoint of wound care, we have

never considered tanning to conform to the principles necessary for first-intention healing. In deep second-degree burns there is probably some destruction of viable epithelial remnants by the tanning. But the greatest violation of surgical principles by tanning is the locking-in of micro-organisms. Most tanned burns, in our experience, eventually become infected.

Local Treatment by Antiseptics.—The production of an eschar by various dyes, designed to prevent the infection so commonly encountered in tannic acid treatment, has the same objections as tanning. Infection is usually not prevented.

Saline Baths or Compresses.—This method is, perhaps, of the greatest use in the late treatment of extensive granulating areas. It has been employed in primary treatment of burns. It is of most use for burns of the hands and face. But in third-degree burns the end-result is a granulating wound which is frequently more or less infected. In our experience, it is quite arduous for an acutely ill patient, and must oftentimes be discontinued because of exhaustion. Its main advantage over tanning is that third-degree areas are ordinarily ready for grafting at a considerably earlier date.

The Closed Compression Dressing Method.—This treatment is based on sound surgical principles. It is in essence the Orr-Trueta treatment of compound fractures applied to burns. It was originated by Koch and Mason, and Mason[1] states that by this method the mortality rate in the Children's Wards, at Cook County Hospital, has been reduced from 10 to 3.9 per cent. In brief it consists in: (1) Thorough soap cleansing. (2) Débridement of loosened epidermis. (3) The application of ointment-saturated gauze strips over the area. (4) The application of a voluminous dressing, applied under moderate pressure. (5) Leaving the dressing in place for 10 to 14 days.

We are in complete accord with this method, and believe it superior to any other now in use. It is ideal for first- and second-degree burns. Healing *per primam* usually occurs in two weeks. The patient is more comfortable than by any other method about which we know, and there is a minimum expenditure of effort in nursing care.

However, when third-degree burns are present, it is not ideal. The cleansing and superficial débridement does not remove all the dead tissue. This remains as a nidus for infection and a focus of absorption. These areas must, of necessity, become granulating wounds, with their attendant problems.

Immediate Skin Grafting.—In an attempt to fulfil the principles for wound healing *per primam,* we have débrided the third-degree areas, and immediately covered them with split-thickness skin grafts.

Our plan of treatment at present is as follows:

1. To be acceptable for immediate débridement and skin grafting, the burn must have occurred not more than six hours previously. With experience it may be possible to extend this time limit.
2. Hematocrit and plasma protein determinations are made at once.
3. Parenteral plasma is started.

4. Under anesthesia, the burn is washed with soap and soft gauze for ten minutes. This removes the blisters and desquamated epidermis from the second-degree areas. The cleansing is completed with a saline flush. No antiseptic is used.

5. The blood pressure and pulse is carefully watched for any signs of incipient shock, and the plasma administration is governed accordingly.

6. Obvious third-degree areas are completely excised, and hemostasis carefully attended to. If there is doubt about an area being third-degree in depth, it is not excised, as it is in these questionable areas that hair follicles and glandular remnants are usually present in sufficient amounts to allow spontaneous epithelization.

7. Grafts of about 0.010 of an inch thickness are cut with the dermatone and sutured over the excised areas.

8. The donor areas are dressed with one thickness of vaselined gauze smoothly applied, and a snug, sterile gauze-roll bandage.

9. The entire burned area is dressed in the same manner. A voluminous gauze dressing is applied over the vaselined gauze and this is held in place under some compression by either Ace-bandage or stockinette applied as a roller-bandage.

10. Adjacent joints are immobilized by the application of plaster encasement directly over the dressing.

11. Plasma, saline, or whole blood is administered during the postoperative period, as indicated by frequent hematocrit, plasma protein, and Hb determinations.

12. The dressing is not disturbed for 14 days unless a mounting fever or other signs indicate that infection has occurred.

13. If the area is healed on removal of the first dressing, a second dressing of vaselined gauze, a thin layer of dry gauze, and an elastic roller-bandage is applied. This is worn as protection for another 14 days. It is then removed and treatment terminated.

This method of treatment and the postoperative course is illustrated in the following case report:

Case 3.—S.M.H., No. 188918; D. B., white, male, age 11, was admitted to Strong Memorial Hospital, one-half hour after being burned on the back. He was not in shock —B.P. 150/85, pulse 100. He was in considerable pain. The burn, as estimated by Berkow's charts, covered about 8 to 10 per cent of the body. It involved the back and a small area over the posterior aspect of the left upper arm. Cold cream had been applied to the burned area.

In the central portion of the burn on the back the skin was an ivory-gray color. About this there was a wide zone of hyperemia on which there were large, weeping areas, intact blebs, and hanging shreds of epidermis. An hematocrit reading, 45 minutes after injury, was 49.3, and the plasma proteins 6.58. He was treated by the method outlined above. During operation, and the immediate postoperative period, he was given 600 cc. of whole plasma and 250 cc. of saline. No shock occurred. His postoperative course was satisfactory. The temperature never rose above 38.7°C. (101.5°F.), and returned to normal on the seventh postoperative day. From the fifth postoperative day

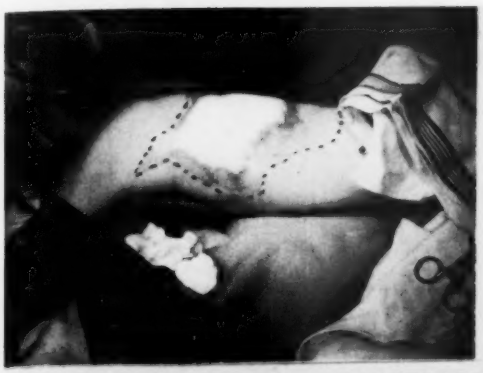


FIG. 1a

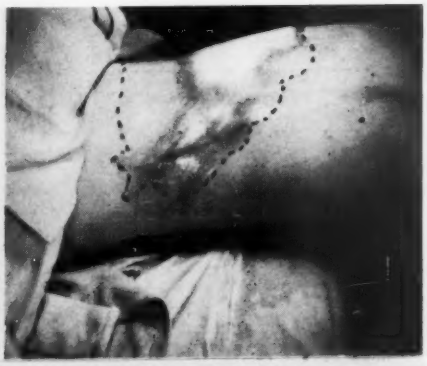


FIG. 1b

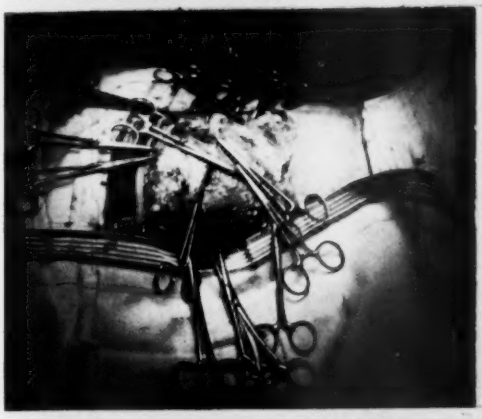


FIG. 2a

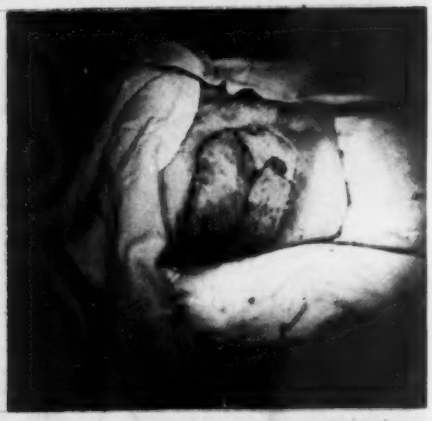


FIG. 2b

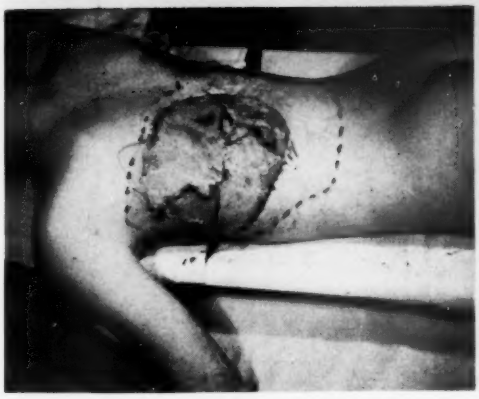


FIG. 3a

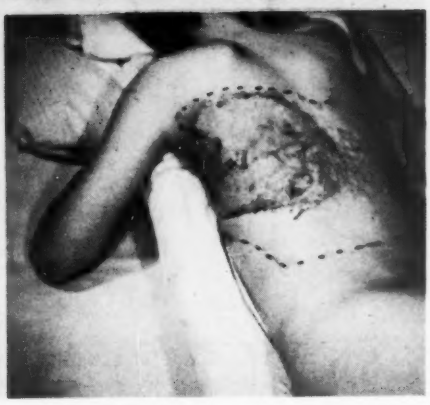


FIG. 3b

FIG. 1.—Case 3: One and one-half hours after injury. Soap cleansing has just been completed, under anesthesia. The extent of the burn is indicated by the dotted area. The white central area is deep third-degree burn. Note that cleansing has completely removed all blisters and desquamated epidermis.

FIG. 2.—Case 3: (a) The third-degree area has been completely excised; in places this excision went down to muscle before normal tissue was encountered. (b) Skin grafts .010 inches thick, cut from buttock and thigh and sutured over excised area.

FIG. 3.—Case 3: Appearance of wound on first change of dressing on the 14th postoperative day. The first- and second-degree burns have healed and the graft has taken completely. The wound has healed *per primam*. He felt perfectly well and was allowed up. Discharged on 17th postoperative day.

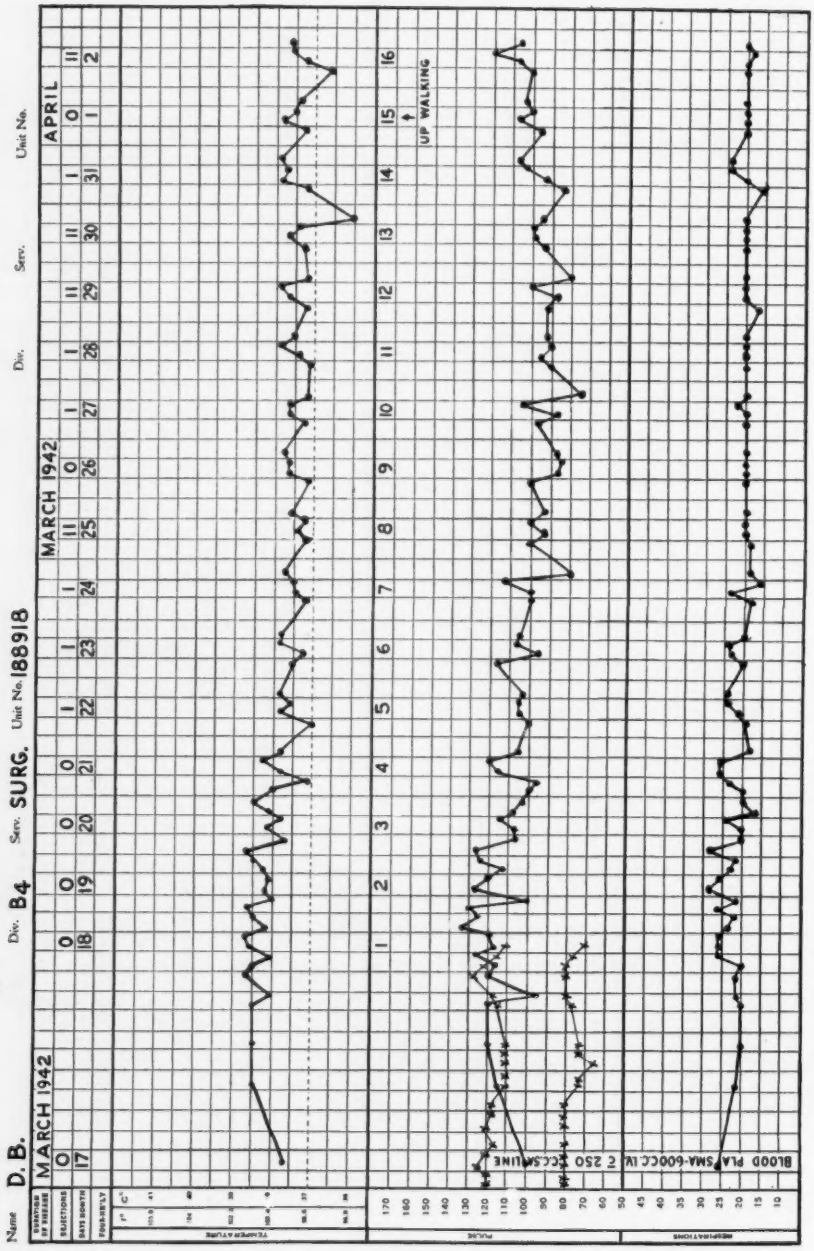


CHART 1.—Case 3: Showing that vital signs returned to normal on fifth postoperative day.

on he was comfortable. The dressing was removed on the fourteenth postoperative day. The entire area was epithelized. One small mixed area of third- and deep second-degree burn, which was purposely not débrided, showed the papillary layer, and was sensitive to touch and temperature changes. The remainder of the burn was painless. A new dressing was applied as outlined above. He was allowed up. On the seventeenth postoperative day he was discharged.

TABLE I

TABULATION OF BLOOD STUDIES IN CASE 3 DURING THE INITIAL TREATMENT AND POSTOPERATIVE COURSE. WITHIN 36 HOURS THE VARIOUS DETERMINATIONS WERE WITHIN NORMAL LIMITS.

Name: D. B.

Date	Time	Specific Gravity	Hemato-crit	Total Protein	Albumen	Glob.	Hb. Gm.	W. B. C.	Remarks
3-16-42	6:45 P.M.	1.0272	49.3	6.58	4.13	2.45			45 min. after burns, before any treatment
	8:40 P.M.	1.0266	47.2	6.19	3.84	2.35			After 100 cc. whole plasma and cleansing and débridement
	9:30 P.M.	1.0270	47.1	5.98	3.90	2.08			After 150 cc. whole plasma during skin grafting
	11:45 P.M.	1.0264	44.7	6.16	4.05	2.11			After 300 cc. whole plasma and skin grafting
3-17-42	4:00 P.M.	1.0256	36.8	5.99	3.86	2.13	11.7	10,000	After 600 cc. whole plasma plus 250 cc. saline, i. v., disc. at 4 P. M.
	9:00 A.M.	1.0263	33.8	6.20	4.03	2.17			No more i. v. fluid; taking fluids well p. o.
3-18-42	11:00 A.M.	1.0275	43.3						No parenteral therapy
3-19-42							11.8	8,000	
3-20-42	11:15 A.M.	1.0271	43.2	6.23	3.47	2.76			
3-21-42							11.3	6,500	
3-22-42									
3-23-42							11.5	8,100	
								Total	600 cc. plasma (whole) 250 cc. saline

CONCLUSIONS

1. Burns are traumatic wounds, accompanied by local loss of blood plasma both from the weeping surface and into the tissues. This plasma loss produces shock by depleting the circulating plasma.

2. Ideal treatment of burns should accomplish the following: Restore lost plasma; prevent further plasma loss; prevent absorption of burned tissue; forestall infection; and promote primary healing.

A first- and second-degree burn, given proper care, will heal *per primam*. A third-degree burn, treated by former methods, can heal only by second intention.

4. By the immediate cleansing and *complete* débridement of a burn, with immediate grafting of deeply injured areas, plus the application of the usual compression dressing and immobilization used over free grafts, it is possible to obtain primary healing of the entire wound.

REFERENCE

[1] Mason, Michael L.: Local Treatment of the Burned Area. Surg., Gynec. & Obst., 72, 250, February, 1941.

THE ELECTRO-ENCEPHALOGRAPHIC DIAGNOSIS OF SUBDURAL HEMORRHAGE

HENDRIKUS SJAARDEMA, Ph.D.,
AND
MARK ALBERT GLASER, M.D.
LOS ANGELES, CALIF.

THE CLINICAL DIAGNOSIS AND LOCALIZATION of subdural and extradural hemorrhage are at times impossible. When such hemorrhage is suspected, it is customary to carry out multiple perforator and bur openings to verify the diagnosis.

The electrical activity of the human cortex, as recorded by the electro-encephalogram, may be modified either temporarily or permanently by such cerebral pathology as tumors, diseases, trauma or chemicals.[1-9] Experimentally produced lesions also definitely modify the electrical activity. The electro-encephalographic wave produced by destructive lesions has been reported by Dusser de Barenne and McCulloch,[10] the wave caused by cortical pressure, without destruction of the underlying brain, has been described by Glaser and Sjaardema.[11] In the experimental production of these pressure lesions a characteristic electro-encephalographic wave pattern was produced in rabbits, which consisted of high *beta* waves superimposed upon *delta* waves. Both clinical extradural and subdural hemorrhage, when there is no damage to the underlying brain, closely simulates the experimentally produced pressure lesions.

In the present communication we wish to present three cases of verified subdural hematoma, in which these characteristic pressure wave patterns were present in each instance, and promptly disappeared after removal of the hematoma. These wave patterns, however, seem not to be pathognomonic of subdural hematoma, or even pressure lesions, because they have been found in hydrocephalus, meningioma, tuberculoma, Parkinson's disease, syphilis, and in a few instances of brain trauma, without operable hemorrhage.

CASE REPORTS

Case 1.—R. R., female, age 27, referred by Dr. J. M. Harris, gave a history of a head injury 15 days prior to admission to the hospital. Surgery revealed a right fronto-temporal subdural hematoma. The electro-encephalogram gave the characteristic wave pattern. These waves disappeared 24 hours after operation, and the brain was entirely normal at the end of six months.

On August 16, 1940, the patient fell, striking her head on a stove. She was unconscious approximately 12 hours. Headaches, that were present for approximately one week, were constant, and situated on the right side. In addition, she had nausea and some vomiting.

Physical Examination.—The patient was a well built young woman, weighing 125 pounds, pulse 84, respirations 20, temperature 98.6°F., blood pressure 115/80. Her general physical examination was essentially negative. *Neurologic Examination.*—The

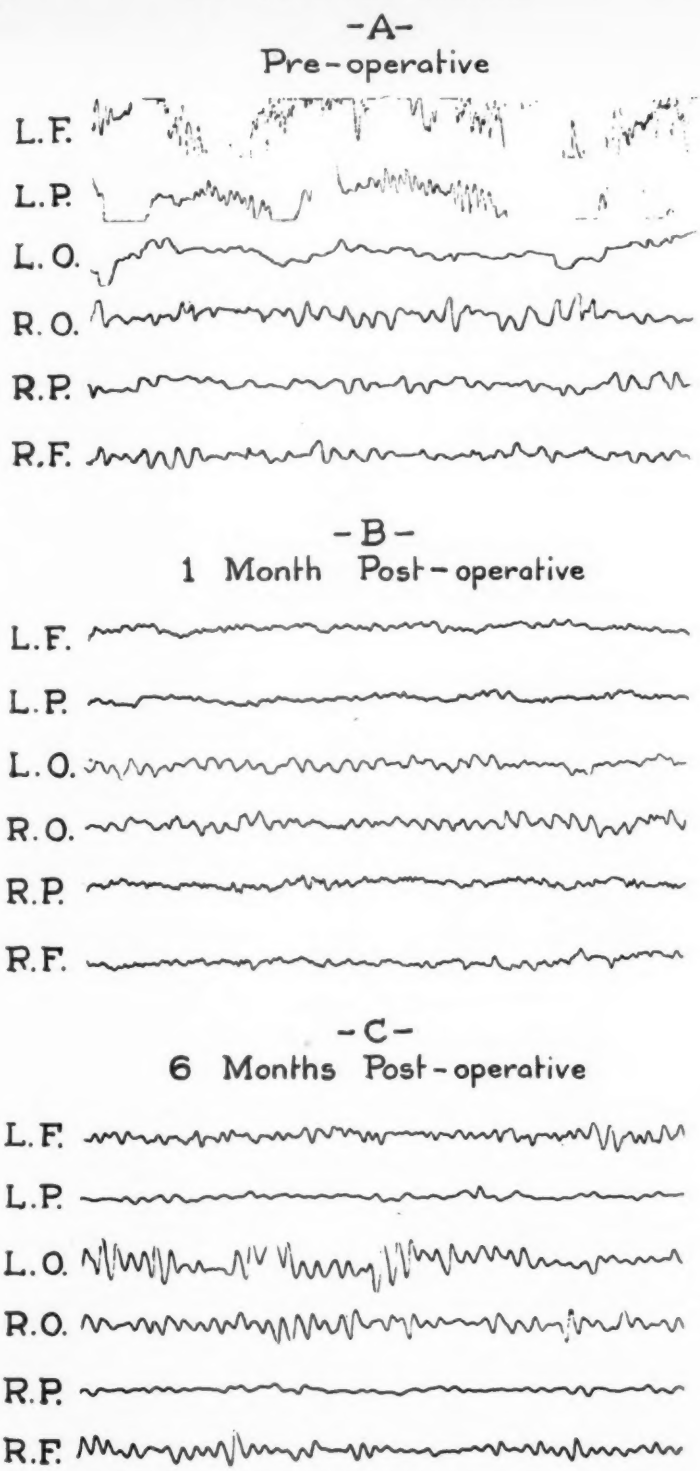


FIG. 1.—Case R. R.: Right Prefrontal Subdural Hematoma. The location of the leads are designated as follows: L. F.—left frontal; L. P.—left parietal; L. O.—left occipital; R. O.—right occipital; R. P.—right parietal; R. F.—right frontal.

A. Note the high voltage *beta* waves present in addition to the *delta* waves in R. F. and R. P.

B. *Delta* waves are still present in R. F.

C. The electro-encephalogram is normal.

pupils were very small, though not pin-point, reacted normally to light and distance, and had to be dilated to visualize her disks. These showed blurring of the margins. The remaining neurologic examination was entirely normal except that the grip in her right hand was 120 compared to 138 in the left hand; and that her reflexes were moderately active in the arms, but hyperactive in the lower extremities. There were no pathologic reflexes. A spinal puncture, on two occasions, revealed a pressure of 550 mm. of water.

The electro-encephalogram revealed a localized area of abnormal electrical activity. This area was located in the right prefrontal region and extended close to the right motor area. The following abnormalities were observed: 1. Slow *delta* waves of approximately one cycle per second, and around 100 microvolts. 2. Rapid waves of about 16 to 25 cycles per second, and from 10 to 20 microvolts. These abnormalities were not present in the corresponding areas of the left hemisphere (Fig. 1A).

Operation.—On the basis of her right-sided headache, localized in the frontoparietal area, the high intracranial pressure, and the characteristic electro-encephalographic waves, exploration was undertaken. The operation was performed under local anesthesia. A skin incision was made in the frontotemporal region, approximately two inches in length. A perforator and bur opening was made in this area. The opening was enlarged with a rongeur. The dura was extremely vascular and exquisitely tender to touch. Cutting the dura caused considerable local pain. When the dura was grasped with forceps an acute sharp pain was referred to the area of the headache and further radiated toward the ear. Beneath the dura was observed the dark-reddish capsule wall, which was extremely soft to palpation. A thin hypodermic needle was introduced through this capsule and one cubic centimeter of brownish-red fluid was withdrawn. The capsule was then opened and about 50 cc. of fluid removed. By careful dissection and washing, this entire structure was removed. The capsule was about ten times as thick as the dura. The underlying brain was compressed by the hematoma in such a way as to leave nearly a half inch space beneath the skull. The cranial opening was approximately in the posterior middle part of this large hematoma, and the capsule extended around this opening in all directions but mostly anteriorly and laterally. The wound was then closed and dressing applied. No drain was inserted. The patient made an uneventful recovery, and was discharged three weeks postoperative.

Subsequent Course.—The electro-encephalogram taken one month after the removal of the subdural hematoma, in the indicated area, revealed some slow *delta* waves in the right frontal region. The *beta* voltage, however, had returned to normal (Fig. 1B). The electro-encephalogram recorded six months postoperative was entirely normal (Fig. 1C).

Case 2.—From the Neurosurgical Service of the Cedars of Lebanon Hospital, March 10, 1940: E. R., male, age 66, had a head injury four months prior to hospital entry. Surgery revealed a left frontoparietal subdural hematoma. Characteristic electro-encephalographic waves were present. The combination waves disappeared 24 hours after operation. Within six months after surgery the *delta* waves disappeared and the electro-encephalogram was entirely normal.

In November, 1939, the patient fell in the bathroom and injured the right side of his head. A large hematoma of the scalp developed. It was not definitely determined whether he had had an unconscious episode and fell or whether he slipped and fell. He remained unconscious for about three minutes and when he recovered the right side of his face was found to be contused and his right thumb pained. He was seen by a physician on 12-19-'39, at which time a drooping of the right side of his mouth was noted, an absence of the right biceps, and of both Achilles reflexes. He also complained of some loss of hearing in the left ear, and weakness of the right grip. The reflexes on the right were more active than those on the left. A spinal fluid examination at this time revealed a total protein of 225 mg. per 100 cc. On 2-26-'40 there was some weakness of

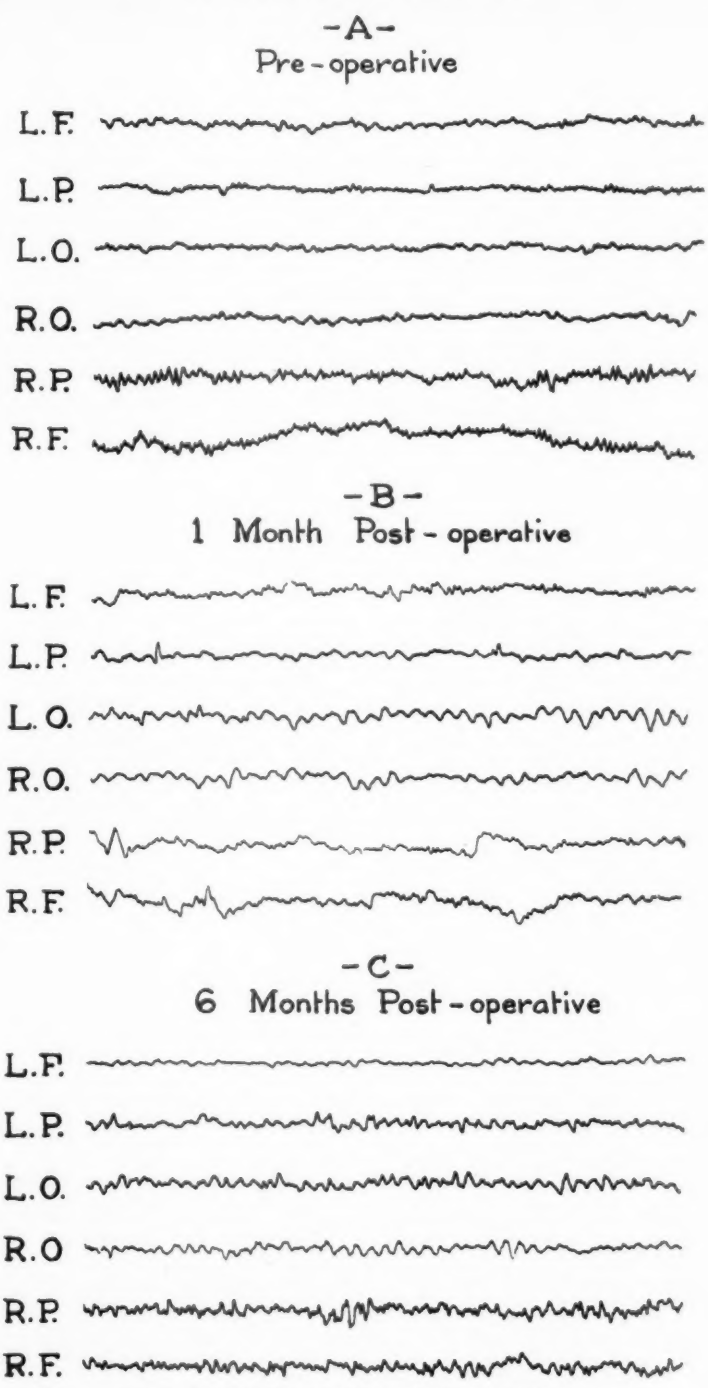


FIG. 2.—Case E. R.: Left Frontoparietal Subdural Hematoma.
A. Combined high *beta* and *delta* waves in L. F., and L. P. Low *deltas* in L. O. and R. O.
B. *Delta* waves in L. F. Remaining leads normal.
C. Electro-encephalogram normal.

his right leg. About March 1st, the weakness of his right leg increased and he developed attacks of dizziness. He had complained of headache since the time of his fall. On March 7th, he developed weakness, unsteadiness and incontinence of urine, as well as periods of mental confusion. On March 10th, he became stuporous, and was admitted to the Neurosurgical Service of the hospital. He was confined to bed, had Cheyne-Stokes respiration, was irrational, pulse 45, blood pressure 130/90. Physical examination was essentially negative.

Neurologic Examination.—There was a definite rigidity of his right arm and leg, associated with weakness. He was irrational but responded at times to questions in a a very slow and confused manner. He was unable to write. The reflexes of the arms were subnormal, the abdominal and cremasterics were absent, and the patellar and Achilles reflexes were subnormal. A spinal puncture revealed a pressure of 225 mm. of water.

On electro-encephalographic examination both occipital areas showed low *delta* in addition to the regular *alpha* waves. The *alpha* voltage was from 50 to 70 microvolts, and the frequency nine cycles per second. The *deltas* were from 50 to 70 microvolts and the frequency around one cycle per second. In the left motor and frontal areas a combination of *delta* and *beta* waves of increased voltage was observed. In this area the *delta* waves were from 75 to 150 microvolts, and about one cycle per second. The *beta* waves were from 15 to 40 microvolts, and from 16 to 25 cycles per second. The right motor area had a smaller amount of *delta* waves, not higher than 75 microvolts (Fig. 2A).

Operation.—Under local anesthesia, a subdural hematoma was found in the left frontoparietal region. The hematoma contained approximately 100 cc. of fluid. After this was removed the underlying brain became visible, and promptly began to pulsate. The brain, however, did not return to the surface of the skull. The entire dissection of the wall of the hematoma was carried out through the perforator and bur opening. Within a few minutes after the hematoma was removed he became quite rational and was able to write his name. He left the operating room in excellent condition.

Subsequent Course.—One month postoperative low *delta* waves were shown in the left frontal area, whereas the other regions showed normal pattern (Fig. 2B). Electro-encephalogram taken six months postoperative was entirely normal (Fig. 2C).

Case 3.—Referred by Dr. L. Gaulden and Dr. G. Esker: R. O., male, age 66, was injured March 11, 1941. The electro-encephalogram showed combination wave patterns. Operation revealed an acute subdural hemorrhage in the right frontalparietal area. The increased *beta* waves disappeared after operation. The patient expired.

This man fell a distance of 12 feet, striking his head, and was rendered unconscious. He had a pulse of 65, respirations 24, temperature 101°F., blood pressure 120/80. There was severe suggillation over the right eye, his nose bled profusely, and he vomited blood. He had a marked contusion over the right frontal region, pupils were dilated, reacted sluggishly to light, and the right pupil was slightly larger than the left. A slight facial paresis existed. There was no evidence of weakness of the extremities. All the reflexes were absent. Spinal puncture revealed a pressure of 400 mm. of water; the fluid was bloody; and showed a count of 60,000 red and 100 white cells per cubic centimeter.

Delta waves, of about one cycle per second and up to 150 microvolts, were present in the right frontal and temporal parietal areas upon electro-encephalographic study. In addition there were high *beta* waves, up to 25 microvolts, and 15 to 25 cycles per second, in this area. The left occipital area showed *delta* waves up to 100 microvolts, and around two cycles per second. The *beta* waves were increased in all areas (Fig. 3).

In view of the high intracranial pressure, the facial weakness, and the characteristic electric waves over the right frontal region, the diagnosis of subdural hematoma, as well as severe brain damage was made.

Operation.—Under novocain anesthesia, an incision was made over the right frontal area, and a perforator and bur opening was made into the skull. The dura was under

tension, somewhat roughened, and vascular. When it was incised a clot oozed out. The skull opening was then enlarged to about the size of a silver dollar. This blood clot extended to the base of the skull, to the edge of the temporal lobe, frontal lobe, and posteriorly as far back as the edge of the ear. It covered the entire hemisphere, and extended to the midline as far as the falx. It was about one and one-half inches thick. By washing, suction, and stripping with cotton and forceps, the entire clot was removed. The original clot must have contained approximately 300 cc. of blood. After it had been removed the brain was found to be markedly depressed and the vessels over the cortex dilated. In addition, the dura was extremely vascular. The roughness and vascularity of the dura prolonged the operation considerably because of the continuous oozing of blood. The wound was then closed.

Postoperative Course.—A plasma transfusion was administered immediately and a blood transfusion later in the evening. The patient's condition postoperatively was not particularly bad, and his pulse and blood pressure again returned to normal. During the night he did fairly well. About 3:00 A.M. his respirations began to change, and his blood pressure became lower. About 6:00 A.M. his condition was poor and it was quite evident he would not survive. The dressing was then opened and no evidence of bleeding was found, air, however, issued from the wound with each respiration of the patient, indicating that the fracture had extended through the nasal sinuses. A spinal puncture was performed and the pressure found to be 100. The patient expired, 3-11-'41, at 1:55 P.M.

Autopsy.—Fractures of the upper ribs on the right side; considerable bleeding in the subparietal space; and some bleeding within the thorax was found. When the brain was removed there was no evidence of hemorrhage apparent. At the tip of the frontal lobe could be seen a large laceration which extended to a depth of two inches. The orbital plate was shattered, more on the right than on the left side, with numerous fracture lines running into the ethmoid and sphenoid sinuses, both of these being filled with blood, which accounted for the bloody vomitus that the patient had. Another fracture line chipped off the tip of the spenoidal ridge. A chip of this bone had entered the cavernous sinus. It was the bleeding from this that infiltrated along the base of the skull and the hemispheres. On top of the orbital plate was an area, the size of a twenty-five-cent piece, which was depressed and spiculated upward into the cranial cavity. Some of this bone had entered the tip of the frontal lobe. Upon removal of these spicules, pieces of brain could be seen adhering to them. In addition, there was a fracture of the right pelvis. The tissues in the posterior peritoneal space were markedly contused and there was a considerable amount of hemorrhage along the ureter, bladder and surrounding tissues.

Gross pathology of the brain revealed a small amount of subarachnoid hemorrhage, with staining of the meninges, most marked in the left parietal region. There was a small contusion in the subfrontal region, which measured 2.5 x 1.5 cm. in greatest diameters. There was also a small cortical hemorrhage, 8 mm. in diameter, at the tip of the right frontal lobe. Otherwise no gross lesions of the brain were evident. Specific attention was given to the left occipital lobe, where no grossly visible lesion was evident. A block of tissue, however, was taken from this area for microscopic study, and also from the right frontal lobe in the region of the contusion. Section through the brain at the level of the thalami showed a small bruise of the right hippocampal gyrus as well as the medial cortex of the parietal lobe. The location of these contusions are indicated on the drawings accompanying Figure 3. *Pathologic Diagnoses.—Gross:* 1. Minor subarachnoid hemorrhage, chiefly left parieto-occipital. 2. Right subfrontal contusion. 3. Minor contusion tip of right temporal lobe. 4. Minor contusions of right hippocampal gyrus. 5. Minor contusions of medial aspect of right parietal lobe.

Microscopic.—Section through the left occipital lobe shows some slight thickening of the meninges, which is not uniform, and which shows no evidence of focal proliferation. There are no observed architectural alterations. The large nerve cells, however, stain poorly. Some of them show acute alteration in the form of acute degenerative changes and a number of them show fatty degeneration. This degeneration is evidently a chronic process and bears no relationship to any recent change. These chronically altered cells however, show these acute changes in the form of loss of Nissl's substance and actual degenerative changes in the cell body. Otherwise, no conspicuous alterations were to be found.

Section from the right occipital lobe shows no essential alterations other than the fatty change in the large nerve cells, which in this section seem less profoundly altered as compared with the left side. The Nissl's substance in these cells is partly retained.

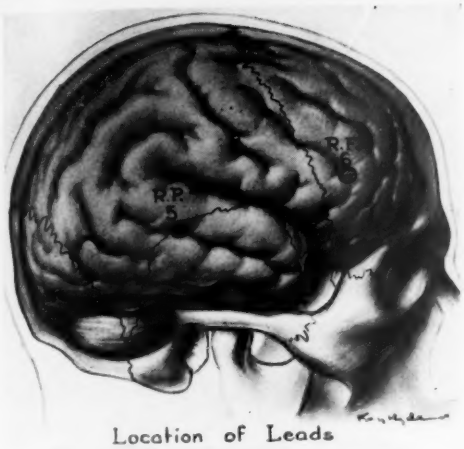
Section taken through the contused area in the right subfrontal region shows a pallor of the affected tissue and in this pale area a few small petechial hemorrhages are found. This contusion involves both the cortex and subcortex over a considerable area and extends fairly deeply into the white matter. *Microscopic Diagnosis:* 1. Subfrontal contusion, right. 2. Acute degenerative changes in the nerve cells of the left occipital lobe.

Twenty-four hours postoperative, *delta* waves around 150 microvolts, two to three cycles per second, were found in all areas. The increased *beta* waves had disappeared (Fig. 3B).

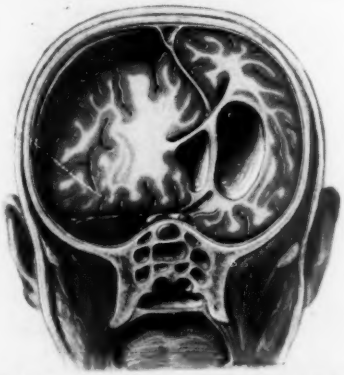
Discussion.—In this particular case we not only had a subdural hematoma situated in the right frontal area but we also had a great amount of brain damage as evidence by the *delta* waves in the other regions of the brain. In spite of the relief of the subdural hematoma by surgery, and its removal, the brain damage was of such severity that the patient expired. We, however, continued recording the waves during the patient's life. These waves continued about 40 seconds after respirations had ceased and the heart action was not audible. The electro-encephalographic recording at this time demonstrated random *delta* waves, and waves of six cycles per second in all areas. The voltage of these waves gradually decreased until the galvanometer did not show any deflexion (Fig. 3C). The patient expired at this time.

In these three clinical cases, in which a subdural hematoma was verified by operation, a combination wave pattern was found. In all of the cases, it disappeared after surgical removal of the lesion. This wave pattern consisted of: 1. *Delta* waves of around one to two cycle waves per second, and 100 microvolts, and up. 2. *Beta* waves alternating or superimposed upon the *deltas* from 16 to 25 cycles per second, and from 10 to 20 microvolts, and up. Although we do not believe this combination pattern is pathognomonic of subdural hematoma, it is noteworthy that we have observed it in cases of hemorrhage wherein a pressure has been applied to the cortex. Taking into consideration the clinical signs, and the association of the combination waves, aids in verifying the existence and localization of the hemorrhage.

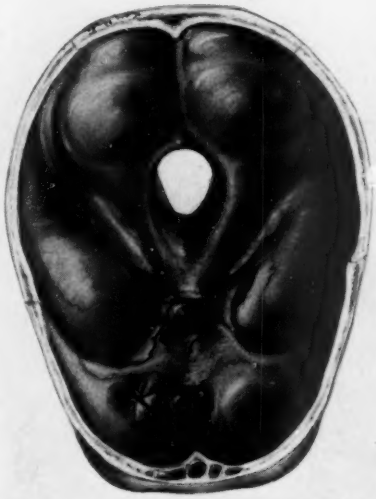
In over 150 cases of head injury in which brain damage was present, and repeated recordings were taken on the same patient, we have found the existence of these combination patterns in only two instances. In none of the entire series was massive hemorrhage suspected. In the two cases mentioned, repeated electro-encephalographic studies showed a disappearance of



Location of Leads



Subdural Hematoma (Schematic)



Depressed Fracture of Base involving Cavernous Sinus

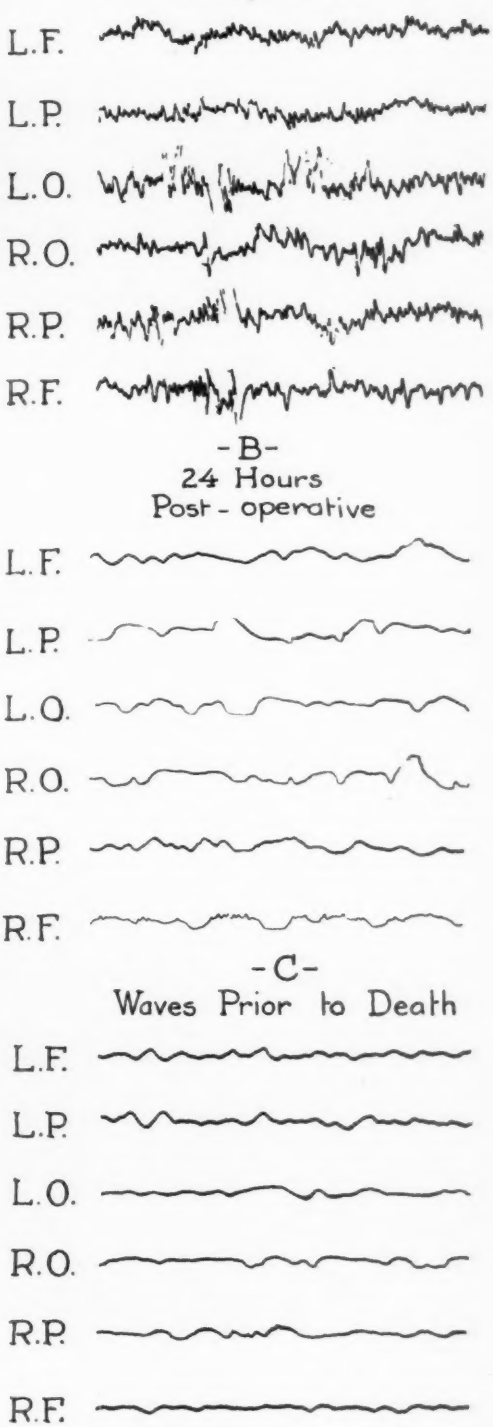


FIG. 3.—Case R. O.: Right Frontoparietal Subdural Hematoma.
A. Combination of high *betas* and *deltas* in R. F. and R. P.
B. *Delta* waves in all areas. Increased *beta* has disappeared.
C. Diminution of electrical activity in all leads.

the combination patterns, and at no time did we have sufficient clinical data to arrive at a diagnosis of large subdural hemorrhage.

CONCLUSIONS

1. In three verified cases of subdural hematoma an electro-encephalographic wave pattern consisting of high voltage *beta* combined with *delta* waves was found, which disappeared when the hemorrhage was removed.

2. This wave pattern is not pathognomonic of subudral hematoma but has been observed in hydrocephalus, meningioma, syphilis, Parkinson's disease, tuberculoma, gumma, and head injuries.

3. The pattern is of value as an adjunct in confirming the diagnosis of subdural or extradural hematoma when the clinical signs and symptoms have indicated such condition to be present.

REFERENCES

[1] Williams, D., and Gibbs, F. A.: Localization of Cerebral Lesions by Electro-encephalography. Tr. Am. Neurol. Assn., **64**, 130-134, 1938.

[2] Walter, W. G.: Electro-encephalography: Aid to Diagnosis. Bristol Med.-Chir. J., **57**, 1-8, 1940.

[3] Case, T. J., and Bucy, P. C.: Localization of Cerebral Lesions by Electro-encephalography. J. Neurophysiol., **1**, 245-261, 1938.

[4] Gibbs, F. A.: Interpretation of the Electro-encephalogram. J. Psychol., **4**, 365-382, 1937.

[5] Gibbs, F. A., Davis, H., and Lennox, W. G.: The Electro-encephalogram in Epilepsy and in Conditions of Impaired Consciousness. Arch. Neurol. & Psychiat., **34**, 1133-1148, 1935.

[6] Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: Epilepsy: Paroxysmal Cerebral Dysrhythmia. Brain, **60**, 377-388, 1937.

[7] Hoagland, H.: On the Mechanism of the "Berger Rhythm" in Normal Man and in General Paretics. Am. J. Physiol., **116**, 77-78, 1936.

[8] Jasper, H. H., Kershman, J., and Elvidge, A.: Electro-encephalographic Studies of Injury to the Head. Arch. Neurol. & Psychiat., **44**, 328-348, 1940.

[9] Glaser, M. A., and Sjaardema, H.: The Value of the Electro-encephalogram in Craniocerebral Injuries. Western Jour. of Surg., Obst. and Gynec., **48**, 689-696, 1940.

[10] Dusser de Barenne, J. G., and McCulloch, W. S.: Some Effects of Laminar Thermocoagulation upon the Local Action Potentials of the Cerebral Cortex of the Monkey. Am. J. Physiol., **114**, 692-694, 1936.

[11] Glaser, Mark Albert, and Sjaardema, Hendrickus: Electro-encephalographic Diagnosis of Extradural and Subdural Hemorrhage. Proc. Soc. Exper. Biol. & Med., **47**, 138-140, 1941.

THE USE OF MYOTOMY IN THE REPAIR OF DIVIDED FLEXOR TENDONS

LESTER BLUM, M.D.

NEW YORK, N. Y.

FROM THE SURGICAL SERVICE OF BEEKMAN HOSPITAL, NEW YORK, N. Y.

DESPITE some notable contributions, the literature of tendon surgery remains persistently optimistic and narrowly technical. While it is the purpose of this report to present still another operative innovation, an attempt will be made to consider its application from the larger viewpoint of hand function.

This can, perhaps, be best done by outlining a general attitude toward the subject of repair of the divided flexor tendon. Let us discard our ambiguous notions of percentages of good or bad results in favor of an analysis of the individual case in terms of controllable and uncontrollable factors.

Among the latter we must list these: The physical nature of the wound; its location in the hand; the degree and nature of contamination; the amount of bruising of the tissues; the extent of stripping of the tendon sheath, and the time-interval before treatment and the presence of complicating fractures. These are the readily recognizable elements that go far to determine an end-result before an instrument has been introduced into the wound.

In addition, there are two other less apparent characteristics of each case that may weigh quite heavily in the final balance. The first is the construction of the injured hand. Does it resemble the pulpy, calloused hand of the laborer, or the dextrous, trim fingers of the artist? The second is the personality of the patient. Is he cooperative and assiduously helping along the restoration of his hand to normal, or does he assume a negativistic attitude and sullenly refuse to move the affected fingers between treatments? There is literally a physical and mental diathesis in each patient which has much to do with the outcome. A willing patient cannot spontaneously dissolve a gallstone or absorb a chest full of pus, but he can work wonders over a period of months with a stiff finger whose flexor tendons may have seemed to be sheathed in very dense adhesions.

While certain technical expedients may serve to ameliorate the untoward effect of any of these uncontrollable factors, they remain as major determinants in the fate of any case regardless of the details of treatment.

The controllable factors are those that become manifest after the patient entered the hospital. They are: The decision for immediate or delayed tenorraphy; the use of a tourniquet; the employment of a proper technic as regards exposure, suture material, and handling of the tissues; and the assignment of one interested member of the staff to the care of every case from reception to final discharge. The importance of each of these

has been repeatedly emphasized by Koch and Mason,[1] Bunnell,[2] and Mayer,[3] so that any further discussion here would indeed be superfluous.

In short, this method of analysis demonstrates the futility of applying the statistical method to a series of tendon cases. Even if the number were extremely large, the many variations of uncontrollable factors alone would render impossible the selection of similar samples in the evaluation of any proposed method or technical maneuver. Furthermore, between the excellent result of complete return of function and the extreme typified by the useless, flopping finger are intermediate grades of recovery to which the ambiguous terms good, fair, indifferent and poor are variously applied.

To make matters more complicated, the fingers are not of equal functional importance since they decrease in usefulness from the index to the fifth fingers. Also, the essential joint of the thumb is the metacarpophalangeal, while that of the other fingers is the proximal interphalangeal joint, a fact of the greatest importance which is commonly overlooked. It is possible to have a satisfactory finger with poorly functioning terminal phalangeal and metacarpophalangeal joints as long as the proximal interphalangeal joint is normal. However, if the motion of the latter is impaired as a sequela of tendon injury, the finger is proportionately crippled.

Thus, the nature of this subject is such that it is as difficult to appraise one's own results as it is to criticise those of others. It is for this basic reason that the method of clinical exposition has been chosen to present the procedure of myotomy as an auxiliary to tendon repair.

Myotomy is designed to control an hitherto uncontrollable factor, *i.e.*, muscle tension on the divided tendon. This exerts its influence from the moment of injury throughout the period of convalescence. At the time of the trauma, it causes the characteristic separation of the tendon ends. There is varying difficulty as the proximal end is found, seized and brought down into the wound for suture. During the first month of convalescence, this muscle tension tends to disrupt the site of repair by its continuous dehiscent effect. It is only after the first three to four weeks, when the tendon is sufficiently healed, that this adverse effect is transformed into a beneficial one, pulling on the tendon in the sheath and so tending to elongate and resolve the adhesions.

Ordinarily, the only means taken to minimize muscle tension is the application of a moulded plaster splint immediately after tenorraphy, with the wrist and fingers in acute flexion. While this rests the muscle and does decrease its tension, it far from obviates the pull at the suture site. This fact is amply demonstrated by the very real tension invariably found as divided flexor tendon ends are being brought together with the patient under complete anesthesia. Despite the immobilizing splint, normal muscle tone exerts its continuous dehiscent force on the suture line during the post-operative period. It is this hitherto uncontrollable factor which has made hazardous the immediate institution of active motion after repair. In fact,

it was just such an unfortunate accident, the rupture of a tendon repaired six days before, that served as the stimulus for this work.

Aside from the original report,[4] there are only two relevant articles. The first by Moser,[5] in 1927, is a short statement to the effect that the retraction of the proximal portion of the divided tendon can be prevented by infiltration of the muscle bellies in the forearm with novocain solution. There are no case reports. The other communication, by McNealy and Lichtenstein,[6] in 1931, is an experimental study, in dogs, of the stages of retraction in a muscle after its tendon has been divided and of the beneficial effect of interrupting the nerve supply.

The rationale of myotomy is, quite simply, the mechanical interruption of the continuity of the tendon with the major portion of the muscle fibers inserting into it. This leaves only those few muscle fibers functioning that are distal to the point of selection, and, therefore, still in continuity with the tendon. This obtains until such time as the myotomy wound has healed and the contractile force of the muscle fibers proximal to the site of myotomy is again transmitted to the tendon.

The flexor muscles of the forearm are of the unipennate and bipennate types with the tendon prolonged throughout the length of the muscle. The muscle fascicles are short and stem from their bony and fascial origins to insert obliquely into the corresponding tendon prolongation which courses along the volar aspect of the muscle to become the tendon in the carpal tunnel and hand. It follows that division of the tendon prolongation results in a loss of contractile power, the degree of which is related to the location of the point of interruption.

The origins of these flexor muscle fibers extend more distally than is shown in the anatomic atlases, so that because of the obliquity of these fascicles, the more distal extend into the carpal tunnel as they unite with the tendon. In fact, the tendons of the flexor digiti quinti are commonly enveloped in muscle tissue as they enter the carpal canal. For this reason, myotomy can be performed just above the wrist and the amount of muscle tissue to be permitted to function is easily measured as it lies exposed in the wound. With each succeeding case, this point of division has progressed distally as confidence in the spontaneous and complete healing of the myotomy wound with full return of muscle strength has grown. In the first case, myotomy was undertaken at about the junction of the distal third with the proximal two-thirds of a flexor muscle belly. In the last cases, only one inch of muscle fibers remained distal to the selected point of division of the tendon prolongation.

The procedure is quite simple and performed in a few minutes. After the divided tendon ends in the hand wound are identified, a short linear incision is made just above the wrist, with clean gloves and instruments. The fascia is divided and then, with an artery forceps, the paratenon tissue is gently separated until the corresponding muscle is identified. This is

rendered easy by gently tugging on the proximal tendon end in the hand wound. The forceps is then passed deep to the muscle delivering it into the wound as shown in the illustration. The point of division of the tendon prolongation is then selected, leaving about one inch of muscle fibers to

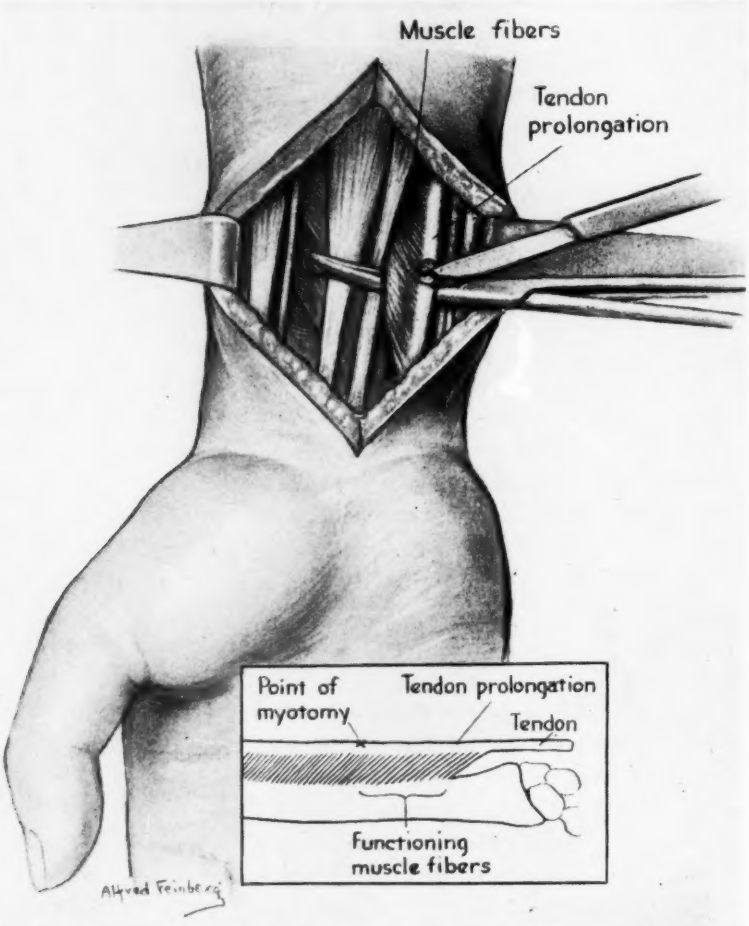


FIG. 1.—The method of dividing tendon prolongation by delivering the muscle into wound after dissecting it free from the enveloping paratenon. Note the distribution of the muscle fibers, particularly as shown in the inset, which is a schematic lateral view at the same level.

function below it. This is labeled "functioning muscle fibers" in the illustration.

As the tendon prolongation is cut, two things can be noticed. The first is the marked diminution of the tension with which the cut tendon ends in the hand can be approximated. The second is the actual transposition of the tendon distally into the hand as the myotomy wound gapes, usually one-half to three-quarters of an inch. The fascia and skin are then closed, and the procedure returns to the hand.

On an empiric basis of a dozen unselected cases, myotomy as an auxiliary to flexor tenorraphy possesses the following characteristics:

1. It is an easily performed, short procedure.
2. It facilitates the handling of the divided tendon ends by eliminating the tension which ordinarily complicates the repair.
3. The danger of disruption of the suture line is obviated. This allows early active motion if so desired.
4. Much finer suture material can be used in the tenorraphy. In the last cases, arterial silk was employed.
5. Because of the ease of approximation of the tendon ends, these can be more completely débrided and trimmed without fear of sacrificing too much tendon.
6. Because of the absence of tension, the finger can be splinted, postoperatively, in extension with only the wrist flexed. This has two advantages. In the first place, it is far easier to regain function in a finger so maintained than in one coiled up in the most acute flexion for three to four weeks. Secondly, in the extended finger the site of tendon suture is withdrawn into the uninjured tendon sheath away from the point at which the sheath, surrounding tissues, and skin were lacerated. These tendons are almost invariably divided when the fingers are in the grasping or acutely flexed position. Thus, the postoperative extension makes for less likelihood of adhesions between the tendon suture site and that of the injury to the sheath and skin.
7. In secondary cases with marked retraction or loss of tendon substances, myotomy renders unnecessary the use of tendon grafts to bridge short intervals. This is demonstrated in the second and tenth case reports below.
8. There has been uniform return of muscle strength in all the cases. As would be expected in so richly vascular a tissue, the myotomy wound evidently heals completely and so allows full restoration of muscle function. This was measured in the first case.

CASE REPORTS

Case 1.—Beekman Hospital No. 33783: J. P., age 43, was admitted, April 18, 1939, shortly after a porcelain faucet handle crumbled in his right hand. He had suffered a short laceration of the palm of the right hand and, on examination, exhibited complete loss of flexion of the interphalangeal joints of the index finger. There was also anesthesia of the radial half of the palm and of the apposing surfaces of the index and middle fingers. Since the criteria for immediate repair were satisfied, operation was immediately performed. The sublimis and profundus tendons to the right index finger were found to be completely divided. With slight extension of the wound it was possible to seize the divided profundus tendon ends. They were approximated under considerable tension. For this reason, with clean gloves and instruments, an incision was made in the lower third of the forearm through which the corresponding profundus muscle belly was identified and isolated. In the lower third of the muscle a short transverse incision was made through the tendon prolongation, resulting in almost complete relief of the tension at the site of injury so that the divided tendon ends, after the clamps holding

them were cut away, were easily approximated with fine Pagenstecher linen thread using the Connell technic. The hand and finger were placed in about ten degrees flexion and a dorsal plaster splint was maintained for 25 days. During this time, guided active motion was allowed each day except on the tenth, when due apparently to edema, no motion was possible. The wounds remained clean. From the fourth week on, radiant heat, massage, and active motion were instituted. The patient returned to work at the end of the fifth week. By the end of the seventh week, there was a normal range of flexion and extension at the interphalangeal joints, with slight restriction at the metacarpophalangeal joints. By the end of the eighth week, he could touch the tip of the finger to the palm. By the twelfth week, the finger could be actively coiled up and moved in all directions with agility equal to that of the normal side. The patient was discharged with a normally functioning finger in the fifteenth week.

Return of muscle power was tested by holding the forearm and finger rigid against a wooden splint and flexing the distal interphalangeal joint over the edge of the splint. At the end of the sixth week, a four-pound weight could be easily moved over a pulley by the terminal phalanx throughout the full arc of flexion. By the twelfth week, an eight-pound weight was easily so moved; by the fourteenth week, nine pounds. Control for the terminal phalanx of the normal index finger was eleven pounds flexion.

Case 2.—Beekman Hospital No. 36924: E. S., age 51, was admitted, December 11, 1940, 23 days after a porcelain faucet handle had crumbled in his right hand, resulting in a wound of the palm which was sutured by a local physician. He came to the clinic for after-care, and it was noticed that there was loss of flexion of the interphalangeal joints of the right index finger and right thumb as well as a loss of sensation along the lateral surface of the index finger and of the web space between it and the thumb. At operation, exposure was obtained by incision along the thenar flexion crease. With considerable difficulty the divided flexor tendon ends of the index finger were dissected free of the scar tissue. The distal end of the flexor pollicis tendon was identified, but it was evident that the proximal end of this flexor pollicis tendon had retracted within the carpal canal where it was coiled back on itself and closely adherent to the other tendons. An incision was made above the wrist for myotomy as well as to assist in the freeing of this proximal portion of the flexor pollicis. However, it was impossible to dissect it free from above either. Since the alternatives of dividing the transverse carpal ligament or of exerting more force and so traumatizing the other tendons and their sheaths were out of the question, the following procedure was performed: The proximal portion of the index sublimis tendon was sutured to the distal end of the index profundus tendon. The proximal end of the index profundus tendon was then swung over to be sutured to the distal end of the flexor pollicis. Because of the tension and the presence of a gap of at least half an inch, myotomy was done on the index profundus tendon. This made possible the suture as described above. Arterial silk was used to unite the tendons and to restore the sheaths as much as possible. Sensory nerve ends in the scar were dissected out and sutured. The wrist was then put up in marked flexion with the fingers extended in the natural position with the aid of a posterior plaster splint. Because a staphylococcus was cultured from the wound at the site of the old blood clot, active motion was not allowed until the end of the third week, the splint being maintained for a month. Physiotherapy was then begun and the patient returned to work involving active use of the hand at seven weeks. Improvement continued progressively until the ninth month. There was normal function of the index finger and only slight restriction of motion of the thumb. The strength of the fingers have practically returned to normal.

Case 3.—Beekman Hospital No. 36324: C. F., age 44, was admitted, August 21, 1940, ten minutes after having caught his right hand in a power saw. He suffered a laceration of the volar aspect of the proximal phalanx of the middle finger with the division of one slip of insertion of the sublimis tendon and a complete division of the profundus tendon and also of the digital nerves. There were other injuries which healed

uneventfully and will not be again mentioned. They were: An avulsion of the nail of the thumb with amputation of the tip; and a laceration of the index finger with partial division of the sublimis tendon. Because of the recent and clean nature of the wound, immediate operation was performed. After identification of the cut tendon ends, myotomy was performed above the wrist and the cut tendon was then sutured with four fine silk stitches, two of the Bunnell type, the tendon ends, of course, being trimmed. A dorsal plaster splint was then applied with the wrist and fingers in 10° to 20° flexion. On the eighth day, guided active motion was interrupted because of the appearance of a stitch abscess which was open. The finger was kept quiet for five days, following which, in view of the rapid healing of the wound, motion was again begun. Physiotherapy was started in the fourth week and the patient went back to work in the sixth week.

Improvement continued progressively until, at the sixth month, there was only slight restriction in flexion of the interphalangeal joints, which was sufficiently small to offer the promise of complete function in the future.

Case 4.—Beekman Hospital No. 38380: W. S., age 42, was admitted, August 18, 1941, six days after both flexor tendons of the right middle finger had been divided by a scissors. The divided tendon ends of the flexor profundus were found through a flap incision over the proximal phalanx. Myotomy was then performed above the wrist and the tendon ends of the finger were then easily approximated with arterial silk. A postoperative plaster splint was applied, with the wrist in flexion and the finger extended. Active motion was allowed throughout the postoperative period. On the sixteenth day, there was some inflammation of the finger wound, and on the twentieth day, pus was released from under the skin flap and a drain inserted. A low grade subcutaneous infection with intermittent discharge of silk sutures continued for nine weeks. At the end of five months, there was about 90° motion in the proximal interphalangeal joint, but practically none in the distal joint. This case has obviously formed adhesions due to the infection and will require plastic procedure some time in the future.

Case 5.—Beekman Hospital No. 38474: H. F., age 20, was admitted, August 20, 1941, shortly after dividing the flexor digiti quinti profundus tendon in the middle phalanx of the finger on a sharp piece of metal. There was also a division of the ulnar digital nerve of the finger. Because the wound was dirty, simple skin suture was performed. The patient was discharged. On September 4th, he was readmitted for tenorraphy and neurolysis. An incision was made along the radial side of the middle phalanx, and then across the distal flexion crease. This flap was then dissected free, revealing an extensive disruption of the sheath, with several foreign particles present. The tendon ends were dissected free. The procedure of myotomy through the usual incision in the wrist was then performed with marked release of tension. The divided tendon ends were then easily sutured together. The digital nerve was then dissected free and its ends placed together. Because of the contamination a molded plaster splint was kept on for three weeks postoperatively. The patient resumed work in six weeks and at the end of five months, showed a normal range of motion of the distal interphalangeal joint and of the finger. The only residuum was a nontender scar. Sensation returned.

Case 6.—Beekman Hospital No. 38514: P. S., age 28, was admitted, September 11, 1941, shortly after catching his left thumb in the blade of an automatic saw. The wound extended through the digital nerves, the flexor pollicis tendon, and actually grooved the bone. Because of the minimal contamination, immediate tenorraphy was performed. There was considerable retraction of the divided tendon ends, as is characteristic of the thumb, and it was necessary to enlarge the opening in the sheath. Myotomy was performed, and it was then possible to approximate the tendon ends with arterial silk. Neurolysis was performed. A molded plaster splint was applied postoperatively and maintained for four weeks, with the wrist in flexion and the thumb in extension. Active motion was permitted at each dressing. Because of the contused nature of the skin wound, and because of an inflammatory reaction with sloughing of the tendon suture, physiotherapy was not begun until the end of the tenth week. Improvement has

been slow, so that while there was full strength of the thumb at the end of five months, there was only about 40° of active flexion in the interphalangeal joint. The patient began working after two months.

Case 7.—B. D., age 24, was operated upon by Dr. Myron Sallick, of New York City, January 1, 1941. On that day he had cut his right thumb with a razor blade and had been unable to flex it since. For proper exposure, the tendon ends were identified, and myotomy was then performed above the wrist. The divided tendon ends were then approximated with fine silk sutures. A posterior plaster splint was applied with the wrist in flexion but with the thumb not otherwise immobilized. Convalescence was uneventful and at follow-up three months later, there was a complete return of function and the muscle strength was equal to that of the normal thumb.

Case 8.—J. L., age five, was operated upon, June 27th, by Dr. Myron Sallick, because of a divided flexor tendon of the right thumb. The accident occurred two hours before admission and was due to a broken milk bottle. The wound was débrided and the tendon sheath was found to be widely lacerated. The capsule of the metacarpophalangeal joint was opened. On approximating the tendon ends, marked tension resulted. Myotomy was then performed above the wrist with marked relief of the tension. The tendon ends were then resutured with fine silk. Five grams of sulfanilamide were placed in the wound. A posterior plaster splint was then applied with the wrist in semiflexion, the thumb being left in a neutral position. Despite a gross infection of the wound with a discharge of pus and early removal of the sutures, active motion was present at the end of the third week. The patient was then discharged to the Out-Patient Department, but failed to return.

Case 9.—Beekman Hospital No. 36013: B. F., age 30, suffered a division of the left flexor pollicis longus tendon when cut by a broken glass jar. The digital nerves were also divided. After the tendon ends were isolated, myotomy was performed and tenorraphy with arterial silk was then easily done. The patient did well, but a secondary contracture due to scar tissue developed. This was not surprising in view of the jagged, contused nature of the original laceration, and the involvement of all the deep tissues, including the capsule of the metacarpophalangeal joint. A secondary plastic procedure was carried out to relieve the contracture. The result was unsatisfactory, since the movement of the thumb is somewhat restricted, despite the fact that the tendon is well healed and causes flexion with normal strength.

Case 10.—Beekman Hospital No. 34090: P. C., age 44, was admitted June 13, 1939, shortly after a water bottle crumbled in his left hand. He suffered jagged lacerations of the ring and little fingers. There was no loss of sensation but there was complete loss of flexion of the interphalangeal joints of the little finger. At operation, only the divided profundus tendon ends were seized and brought together at the middle phalanx. As is characteristic of this finger, there was a good deal of tension. For this reason, an incision was made in the forearm just lateral to the ulnar vessels and nerves. The flexor profundus digiti quinti muscle belly was readily identified. About half of the thickness of the muscle was divided resulting in a definite diminution of tension with much easier approximation of the tendon ends. The myotomy wound gaped more than half an inch. The tendon ends were repaired with a fine linen Connell suture. A posterior molded plaster splint was applied with the hand and finger in slight flexion. This was maintained for four weeks. During the second and third weeks there was a loss of the undercut skin overlying the suture site. The mucopurpulent discharge from this point was evidently due to sloughing skin and not to a real infection. During the fifth week, there were few degrees of flexion of the terminal phalanx and about ten degrees of the proximal interphalangeal joints. Improvement was slow but progressive, so that at the end of ten months, the range of motion at the proximal interphalangeal joint was about two-thirds normal, and at the distal joint, about one-third normal. The result in this case was adversely affected by the loss of the skin flap due to the nature of the trauma.

Case 11.—H. R., age 15 months, was admitted to the care of Dr. Lester Breidenbach, of New York City, because of a history of inability to flex the left, index, and middle fingers, following a deep laceration of the hand, occurring two months before. Exploratory operation revealed a division of the flexor tendons of the affected fingers. After a most difficult dissection, the ends were identified. Myotomy was then performed above the wrist with a gratifying result, so that the tendons in the hand were fairly easily approximated. Postoperatively, a molded plaster splint was maintained for three weeks. Follow-up at four months, showed an excellent result.

CONCLUSIONS

The adverse effects of muscle tension acting upon the site of tendon repair can be obviated by simultaneous division of the corresponding tendon prolongation in the distal forearm.

The procedure of myotomy is easily and quickly performed, and expedites repair of the divided tendon.

The myotomy wound heals spontaneously and completely, with no residual defect of muscle strength.

REFERENCES

[1] Koch, S. L., and Mason, N. L.: Division of the Nerves and Tendons of the Hand. Surg., Gynec., and Obstet., **56,** 1, January, 1933.

[2] Bunnell, S.: Reconstructive Surgery of the Hand. Surg., Gynec., and Obstet., **39,** 259, September, 1924.

[3] Mayer, L.: The Physiological Method of Tendon Transplantation. Surg., Gynec., and Obstet., **33,** 528, November, 1921.

[4] Blum, L.: Partial Myotomy in Treatment of Divided Flexor Tendons of Hand. Annals of Surgery, **113,** 460-463, March, 1941.

[5] Moser, E.: Zur Sehnennaht. Zentralbl., f., Chir., **54,** 1606, June 25, 1927.

[6] McNealy, R. W., and Lichtenstein, M. E.: Muscular Relaxation Produced by Novocain as Aid in Tendon Repair. Surg., Gynec., and Obstet., **53,** 40-45 July, 1931.

BRIEF COMMUNICATIONS

ARTERIOVENOUS FISTULA

CASE REPORT

HAROLD D. CAYLOR, M.D., LEONARD LONG, M.D.,
AND
JACK L. EISAMAN, M.D.
BLUFFTON, IND.
FROM THE CAYLOR-NICKEL CLINIC, BLUFFTON, INDIANA

WE ARE REPORTING a case of traumatic popliteal arteriovenous fistula which occurred in a man, age 20. This lesion was successfully treated by quadruple ligation.[1]

When an arteriovenous fistula involves vessels of considerable size the derangement of cardiac function slowly and progressively increases. As Pemberton[2] observed, the larger the leak the greater the volume of blood that will be shunted and the more serious the consequences. To maintain adequate circulation to the capillaries nature compensates by increasing the systolic pressure and, also, by increasing the total blood volume.[3] Although the heart may carry on for many years under the added load caused by a small arteriovenous fistula the circulatory function becomes progressively more upset. Because of the increased blood flow the artery proximal to the fistula becomes enormously dilated, this, in turn, causes further enlargement of the fistulous opening. Thus, a vicious cycle is established which must inevitably lead to congestive heart failure.[2]

Holman,[4] and Reid and McGuire[5] have noted so-called arterialization of the veins and venification of the arteries. The former is explained by the increased work and adaptation to heightened pressure, and the latter (venification) is noted as thinning, tortuosity and dilatation proximal to the fistula. Because of the fistula and the impaired circulation distal to the opening, intermittent claudication,[6] chronic ulcers,[5] engorged veins, and cyanosis of the involved extremity are the rule. Branham's[5] bradycardia phenomenon (slowing of pulse rate when the fistula is closed) seems directly related to the size of the fistula and the seriousness of the cardiac damage. The arteriovenous aneurysm is a powerful stimulant to the development of collateral circulation in the affected extremity, and it is because of this collateral circulation that portions of the artery and vein can be ligated and excised.[6] In young, growing persons there is an actual lengthening of the extremity due to increased vascularity of the growth centers of the long bones,[6] and this was noted in our case.

Case Report.—G. M., male, age 20, a truck body builder, first presented himself at the Clinic in December, 1936, complaining of a swollen left leg. Five years before, when he was 15 years old, he was sitting on the ground playing with a revolver. He accidently discharged the gun and shot himself in the inner side of the left thigh at about the junction of the lower and middle thirds. Immediately after the shot he

jumped up and ran approximately one-half mile. Then his leg cramped so severely he could run no more. He was carried to a physician's office, and a roentgenogram revealed the bullet lodged in the popliteal space. No attempt was made to remove it. The leg was swollen three or four times its normal size, and it has remained so, more or less, ever since the accident. After a few days the patient was up and about on crutches with his leg in a semiflexed position. Within six weeks he was walking on his toes, and it was several weeks more before he could walk with the heel of the left foot to the ground. Walking a long distance caused cramps in the calf muscles and these ordinarily were relieved if he would sit down and rest.

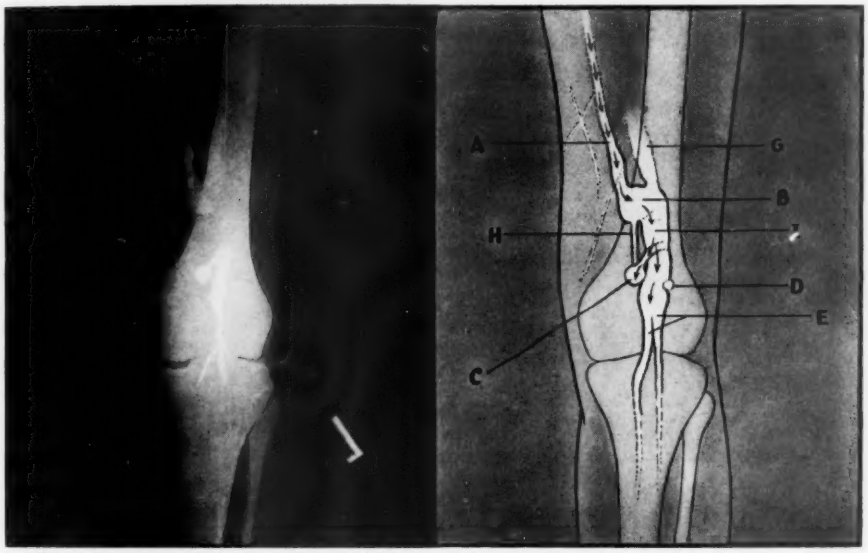


FIG. 1.—Anteroposterior arteriogram taken immediately after injection of thorotrast. Arrows indicate direction of flow of blood through the fistula. A. Popliteal artery above fistula. B. Arteriovenous fistula. C. Communicating vein. D. Bullet. E. Branches of popliteal vein. G. Popliteal vein above the fistula. H. Popliteal artery below the fistula. I. Popliteal vein below the fistula.

Subsequently the patient was enrolled at a C. C. C. camp, and when he was given an hypodermic injection of a vaccine for immunization the left leg swelled considerably more than usual. He reported to the medical officer and was sent to an army post hospital for observation, where he was kept 10 days. Roentgenologic studies were made, and he was returned to the camp for duty. A second hypodermic injection of vaccine was given and again the leg became greatly swollen, and this happened a third time following another injection. This great tumefaction persisted for about a week after the third injection and was not altered by overnight rest in bed or by moderate exercise.

The patient first noticed a thrill on the inner side of the left thigh, four or five centimeters above and below the wound entrance, soon after the accident, and it has persisted. This thrill or bruit was not so noticeable to the patient when we examined him as it had been formerly, but we noticed that a distinct vibration occurred with each cardiac cycle. Since the accident the left foot had been warmer than the right foot. All the superficial veins of the left thigh, leg and foot were more prominent than those of the right and had been since soon after the accident.

Physical Examination.—This revealed a well-nourished young man, 72½ inches tall, weighing 162 pounds. The blood pressure in the left arm was 140/60. Pulse 100, regular; temperature 98.6°F.

The left thigh, measured at a level five centimeters above the patella, was 10.5 cm. greater in circumference than the right. The left leg, measured at a level 20 cm. below

the patella, was 7.7 cm. greater in circumference than the right. The left lower extremity was 2.1 cm. longer than the right.

All the superficial veins of the left leg and thigh were prominent. A thrill was palpable on the inner aspect of the left thigh, extending from the wound of entrance down to the knee, and a bruit was heard in the same area. It was much more difficult to feel the pulsations of the left dorsalis pedis artery than of the contralateral vessel.

Roentgenograms of the left knee revealed a foreign body (bullet) in the popliteal space (Figs. 1D and 2D). Chest roentgenogram showed the cardiac dimensions near the upper limits of normal (Table II, and Fig. 3A).

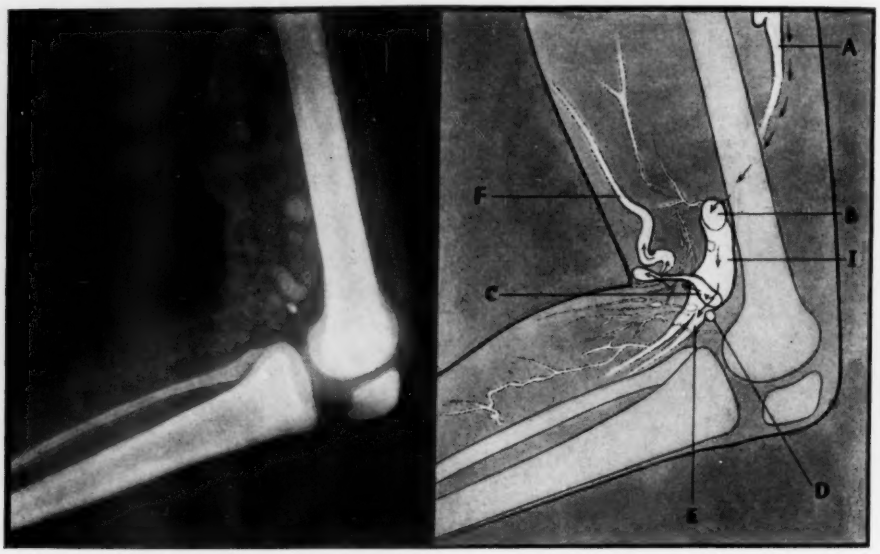


FIG. 2.—Lateral arteriogram taken immediately after Figure 1. Arrows indicate direction of flow of blood through the fistula. A. Femoral artery. B. Arteriovenous fistula. C. Communicating vein. D. Bullet. E. Branches of popliteal vein. F. Superficial vein. I. Popliteal vein below fistula.

A tentative diagnosis of an arteriovenous fistula was made. The gravity of the situation was discussed with the patient and his mother, and it was proposed that further roentgenologic studies with thorotrast injections be made as a guide to proper surgical procedures. Operation was, however, refused, and the patient was not heard from for approximately two years.

On his return most of his symptoms had become aggravated and, in addition, there were two lesions resembling varicose ulcers on the inner surface of the left leg, about four centimeters below the knee. A bruit could still be heard on the inner aspect of the thigh. At this time the blood pressure readings were taken on extremities and are as follows: Left arm 130/60; right arm 130/60; left thigh 140/60; right thigh 130/80. The pulse rate remained 100 per minute. After a blood pressure cuff on the left thigh was inflated sufficiently to close off the aneurysm the pulse rate dropped to 72 per minute within 15 seconds. Thorotrast, (12 cc.), was injected into the left femoral artery, using the methods of Yater[7], and Horton[8], and roentgenograms were taken (Figs. 1 and 2).

Description of Roentgenograms.—The first film was obtained immediately after the injection, with the projection anteroposterior. A lateral view was then exposed as quickly afterward as possible. Digital pressure was maintained on the femoral artery throughout these procedures. In spite of this the thorotrast passed rapidly from the arterial to the venous circulation, so that the two views do not demonstrate entirely

the same vessels. The anteroposterior view shows the fistula itself while in the lateral view the tortuous collateral venous circulation is well shown.

The thorotrast can be traced through the fistula as indicated by the arrows in Figs. 1 and 2. The popliteal artery (Fig. 1A) appears near the inner border of the thigh extending downward and mesially to a level approximately 22 cm. above the joint space where the fistula is seen as a rounded collection of dye (Fig. 1B). After the dye passes through the fistula into the dilated vein it becomes more dilute and the shadow fainter (Fig. 1I). Because of the retrograde flow against resistance the dye concentrates lower down in the popliteal vein, which is particularly well shown with its two branches (Fig. 1E). The bullet is seen lodged against the lateral wall of this vein (Figs. 1D and 2D). The thorotrast then passes through the dilated communicating branches (Figs.

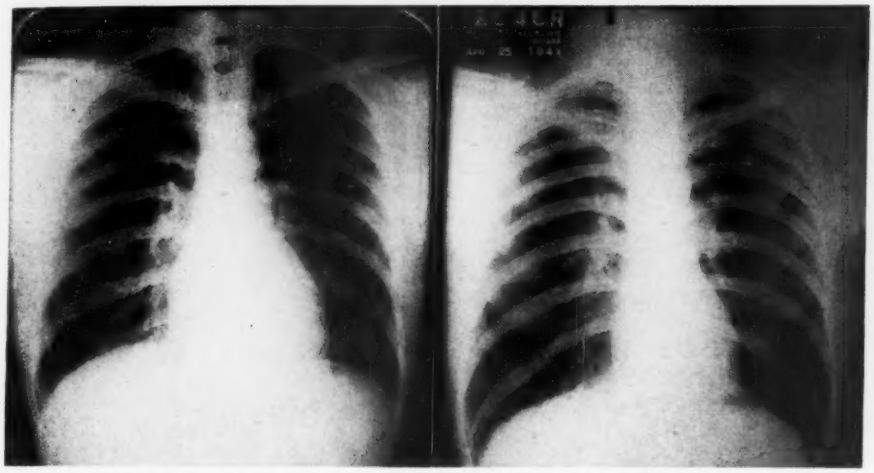


FIG. 3.—A. Chest roentgenogram taken before operation. Area of heart and great vessels 180 sq. cm. B. Chest roentgenogram taken three years after closure of the fistula. Area of heart and great vessels 135 sq. cm.

1C and 2C) to the superficial veins (Fig. 2F). The popliteal artery distal to the fistula is barely perceptible as a narrow streak of dye (Fig. 1H) because the blood flow is predominantly through the fistula and in a retrograde direction down the vein.

Laboratory Data.—Electrocardiogram: Pulse rate 100; sinus tachycardia; moderate right axis deviation. Urine: Negative. Blood: Hemoglobin (S) 93 per cent; erythrocytes 4,500,000; leukocytes 14,000. Kline: Negative. Temperature studies of the extremities with the thermocouple showed a higher temperature in the left thigh, leg, and dorsum of the foot and lower temperature in the left metatarsal area as compared with the right (Table I).

Operation was again offered and was accepted by the patient and his family.

Operation.—February 26, 1938, at the Wells County Hospital. Under ethylene, a longitudinal skin incision was made over the popliteal space just lateral to the semimembranosus and semitendinosus muscles. The superficial fascia was incised and the dissection carried down to the aneurysm, which measured approximately 3.5 cm. in diameter and was in the proximal part of the popliteal space. The artery was isolated from the opening in the adductors above the aneurysm and down through the popliteal space beyond the aneurysm. The popliteal artery distal to the aneurysm was smaller than proximal to it (0.3 cm. distal and 0.5 cm. proximal). The popliteal vein was greatly dilated distal to the fistula, and the stream of blood from the opening between the artery and vein could be plainly seen through the vein wall going in a retrograde direction down the vein. The popliteal vein proximal to the fistula was 1 cm. and distal it was 2.0 to 2.5 cm. in diameter. The popliteal vein and artery lay side by side with

the aneurysm protruding posteriorly. Pressure on the proximal popliteal artery stopped the purr over the aneurysm. The fistulous opening was approximately 0.5 cm. in diameter. All four vascular limbs of the aneurysm were doubly ligated with No. 3 chromic catgut after the technic described by Horsley and Bigger.[1] Fascia was closed with No. 1 plain catgut and a rubber dam drain was inserted down to the fistula. The skin was closed with silk.

TABLE I

TEMPERATURE STUDIES WITH THERMOCOUPLE*

Area	Before Operation		3 Years After Operation	
	Left	Right	Left	Right
Palm	94.5° F.	95.5° F.		
Thigh	94.8° F.	94.0° F.	95.0° F.	94.5° F.
Leg	96.5° F.	93.8° F.	96.0° F.	96.0° F.
Dorsum of foot	86.0° F.	85.8° F.	91.0° F.	90.0° F.
Metatarsal	78.3° F.	79.2° F.	91.0° F.	91.0° F.

* All readings taken at analogous points on each extremity.

Postoperative Course.—When the patient entered the hospital his blood pressure was 140/80 and pulse 88. The first postoperative day the blood pressure was 120/80 and pulse 104. On the fourth day the temperature was 98.8°F., pulse 88. The wound was dressed and rubber dam drain removed. At no time in these first four days was there ever any evidence of impaired circulation in the left foot or leg. On the tenth postoperative day the patient was in a wheel chair. The pulse rate varied from 64 to 80. All sutures were removed on the thirteenth day, and the patient was dismissed walking on crutches on the following day.

Subsequent Course.—Three years after the operation the transverse diameter of the heart had diminished 2.8 cm. and the aorta 0.4 cm. The area of the heart and great vessels was decreased by 45 sq. cm. (Table II and Fig. 3). At this time roentgenograms showed increased density of the spleen from thorotrast contained in it.

TABLE II

CARDIAC MEASUREMENTS

	Before Operation	After Operation
Transverse diameter of heart	13.6 cm.	10.8 cm.
Transverse diameter of chest	29.5 cm.	29.7 cm.
Oblique diameter of heart	14.4 cm.	12.4 cm.
Area of heart and great vessels	180 sq. cm.	135 sq. cm.

TABLE III

OSCILLOMETRIC DETERMINATIONS THREE YEARS AFTER OPERATION

Area	Left	Right
Popliteal area	¾	5
Anterior tibial artery, 25 cm. below patella	1½	5

Oscillometric determinations three years after the operation are indicated in Table III. The deep circulation was almost *nil* in the popliteal and anterior tibial arteries. However, the collateral circulation was good as indicated by thermocouple readings taken at the same time (Table I).

DISCUSSION.—This case illustrates most of the features seen in an arteriovenous fistula occurring early in life. The affected leg was longer than its fellow, larger in circumference, warmer, and exhibited varicose veins and ulcers. The blood pressure and pulse pressure revealed the expected changes of hypertension; low diastolic pressure and large pulse pressure. The heart was enlarged and Branham's bradycardia phenomenon could be demonstrated.

It is important, as others have noted, that a contemplated operation on an arteriovenous fistula should be delayed until an adequate collateral circula-

tion has been established. This can be determined in part by thermocouple studies of skin temperature. If this instrument is not available, histamine (1 or 10,000 solution) will give almost the same information. In this case a double ligation of each limb of the aneurysm was performed, and the patient has now been perfectly well for three years.

REFERENCES

[1] Horsley, J. S., and Bigger, I. A.: Operative Surgery. C. V. Mosby Co., p. 153, 1937.
[2] Pemberton, J. de J.: Discussion, Arteriovenous Fistula. Proc. of Staff Meet. of Mayo Clin., **13**, 804, 1938.
[3] Holman, Emil: Arteriovenous Aneurysm. MacMillan Co., p. 40, 1938.
[4] Holman, Emil: Clinical and Experimental Observations on Arteriovenous Fistula. ANNALS OF SURGERY, **112**, 840, 1940.
[5] Reid, M. R., and McGuire, Johnson: Arteriovenous Aneurysms. ANNALS OF SURGERY, **108**, 643, 1938.
[6] Herrman, L. G., and Reid, M. R.: Management of Arteriovenous Aneurysm in the Extremities. Am. J. Surg., **54**, 17, 1939.
[7] Yater, W. M.: Thorotrast Arteriography of the Extremities: Report of Illustrative and Unusual Cases. Am. Heart. J., **12**, 383, 1936.
[8] Horton, B. T.: Arteriovenous Fistula Involving the Common Femoral Artery Identified by Arteriography. Am. J. M. Sc., **187**, 649, 1934.

SYMPATHETIC DENERVATION LIMITED TO THE BLOOD VESSELS OF THE LEG AND FOOT: II

LAWRENCE N. ATLAS, M.D.

CLEVELAND, OHIO

FROM THE PERIPHERAL VASCULAR SERVICE, DIVISION OF SURGERY, CLEVELAND CITY HOSPITAL; AND FROM THE DEPARTMENTS OF ANATOMY AND SURGERY, SCHOOL OF MEDICINE, WESTERN RESERVE UNIVERSITY

IN A RECENT preliminary report,[1] the anatomic arrangement of the ganglia and rami communicantes of the lumbar sympathetic trunk, as personally observed in the dissecting room, and the surgical application of these observations were presented in considerable detail. The purpose of this communication is to reemphasize, revise, and supplement some of the more important of the originally published observations:

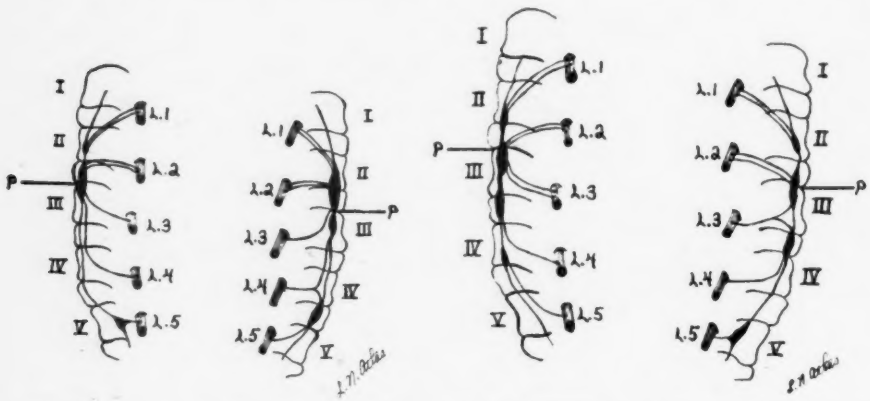


FIG. 1.—Showing some of the typical variations in the topographic anatomy of the lumbar sympathetic trunk. Levels designated by P are the most caudal at which the trunk may be divided without running the risk of obtaining an incomplete denervation of the blood vessels of the leg and foot (ref. Fig. 3.).

(1) Because of erratic fusion of lumbar sympathetic ganglionic tissue, it is impossible to designate lumbar ganglia on a numerical basis with any degree of accuracy.

(2) The only lumbar ganglion which is constant in position and connections is that which straddles the second lumbar intervertebral disk, and which is constantly connected with both the second and third lumbar spinal nerves. (This observation has been confirmed by R. H. Smithwick[2]). Ganglionic tissue connected with the first lumbar spinal nerve may be fused with its rostral pole; and ganglionic tissue connected with the fourth lumbar spinal nerve may be fused with its caudal pole (Fig. 1).

(3) Because of this unpredictable variation in the number and position of the lumbar sympathetic ganglia, it is preferable to describe a lumbar sympathectomy not on the basis of which ganglia were removed, but from the anterolateral surfaces of which vertebral bodies the trunk was resected. This

may be substantiated roentgenographically by placing silver clips on the severed ends of the sympathetic trunk (Fig. 2).

(4) Since preganglionic fibers rarely join the sympathetic trunk below the level of the second lumbar vertebra, the simple maneuver of dividing the trunk at the upper pole of the third lumbar vertebra interrupts the flow

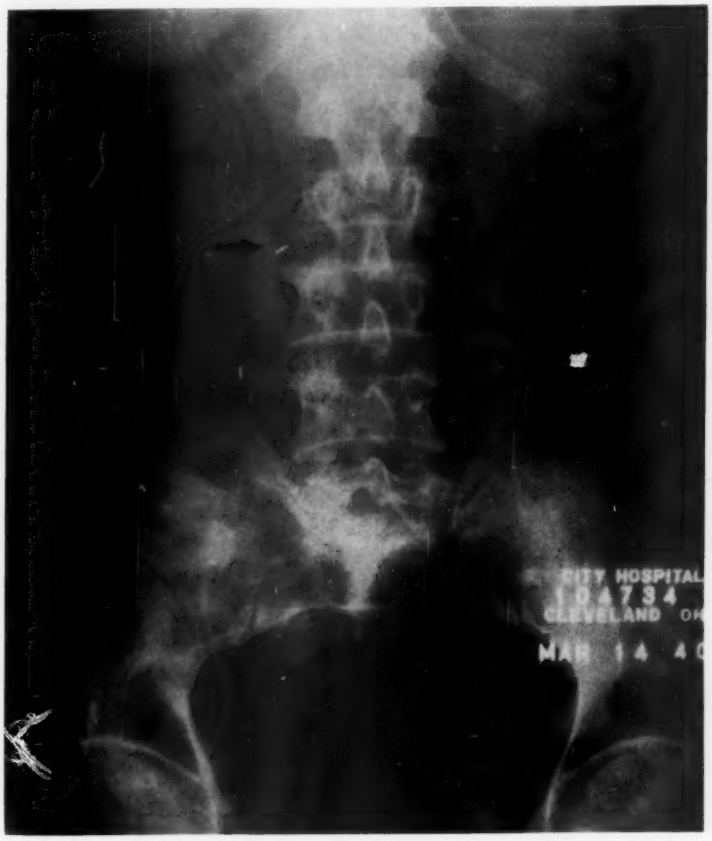


FIG. 2.—Postoperative roentgenogram showing shadows of silver clips on severed ends of lumbar sympathetic trunk (indicated by arrows). Position of upper clip indicates that trunk was severed at level of intervertebral disk between second and third lumbar vertebrae. Lower clip is point at which caudal portion of severed trunk was buried in the psoas muscle after having been stripped from third lumbar vertebra. (Note sacralization of fifth lumbar vertebra).

of vasmotor impulses to those blood vessels of the lower extremities which receive their innervation through the branches of spinal nerves L. 3, 4, 5; S. 1, 2, and 3 (Figs. 1 and 3). The vessels thus denervated include the popliteal artery and its branches and the cutaneous vessels distal to the thigh. To prevent regeneration, the caudal portion of the severed trunk is stripped for a distance of several inches and, as is technically most convenient, either resected or buried deep in the adjacent psoas muscle. In addition to preventing regeneration, this maneuver also destroys any connections of the trunk with the third lumbar spinal nerve, which might have been

missed when the trunk was divided. This is important in those rare instances in which preganglionic impulses reach the sympathetic trunk over a white ramus from the third lumbar spinal nerve.

At the present time, 68 sympathetic denervations, limited to the blood vessels of the foot and leg, have been performed. This limited type of denervation has proved to be quite adequate for the treatment of Raynaud's disease and other vasospastic dystrophies in which the disturbance was limited to the foot and leg. It has also been found to be of value in the treatment of noninflammatory obliterating arterial disease of the lower extremity in which an abnormal degree of peripheral vasoconstrictor tone inhibited the development of a collateral circulation.[3]

In three instances, sweating and vasoconstriction involving the inner border of the lower leg and foot have been observed postoperatively. Roentgenographic check-up of the position of the silver clips revealed that the trunk had been divided at the lower pole of the third lumbar vertebra instead of at the upper pole, thus leaving the postganglionic rami to the third lumbar spinal nerve intact.

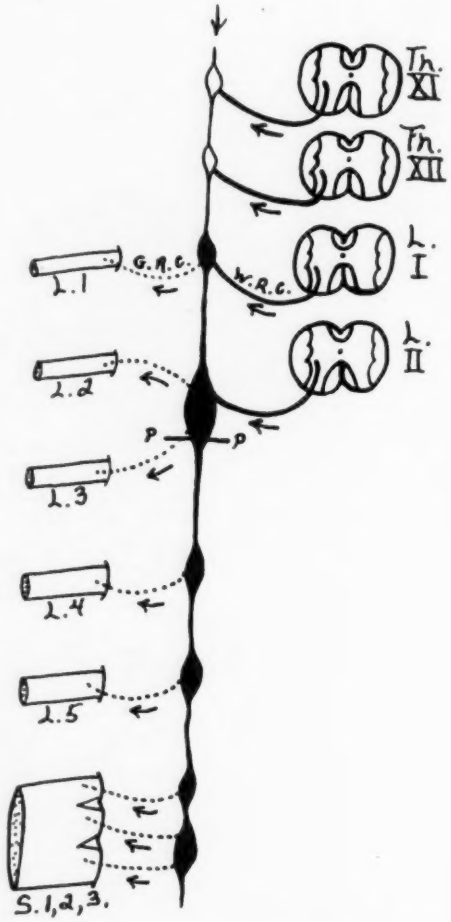


FIG. 3.—Diagrammatic sketch of pre- and postganglionic sympathetic innervation of the blood vessels of the lower extremity. P—P indicates the point at which the flow of vasomotor impulses is interrupted by dividing the sympathetic trunk at the upper pole of the third lumbar vertebra and stripping the caudal portion from the entire length of that vertebra. Dividing the trunk at a lower level leaves the postganglionic ramus to L/3 intact. Branches of this ramus may reach as far distally as the foot.

Arrows indicate direction of vasomotor impulse flow.

W. R. C. = Preganglionic rami.
G. R. C. = Postganglionic rami.

These three cases are considered to be significant because they indicate that sympathetic fibers carried in the branches of the third lumbar spinal nerve may occasionally reach as far distally as the foot. This may explain the empiric observation of Leriche,[4] and others, that resection of the upper end of the lumbar sympathetic trunk often yields a more thorough denervation of the foot than does resection of the caudal portion. If incomplete denervations are to be consistently avoided, the exposure of the sympathetic trunk must certainly be carried at least as high as the intervertebral disk between the second and

third lumbar vertebra before the trunk is divided and stripped (Fig. 1).

The question arises as to how this particular spot is recognized at operation. It is identified by the presence of the large ganglion which is connected with both the second and third lumbar spinal nerves. It is my practice to carry the dissection upwards until this ganglion is exposed, and to sever the trunk through the central portion of the ganglion.

It will undoubtedly be observed by those who have had experience in the field of sympathetic nerve surgery that the technic described is no different from that usually employed. It has not been the purpose of this or the previous communication to offer a new method for denervating the blood vessels of the lower extremity. There are no new methods for accomplishing this end. Rather, the purpose has been to present a clearer appreciation of the precise anatomy of the operation of lumbar sympathectomy.

REFERENCES

[1] Atlas, L. N.: A Modified Form of Lumbar Sympathectomy for Denervating the Blood Vessels of the Leg and Foot: Anatomic Considerations. ANNALS OF SURGERY, **111**, 117, 1940.

[2] Smithwick, R. H.: Personal communication.

[3] Atlas, L. N.: Lumbar Sympathectomy in the Treatment of Selected Cases of Peripheral Arteriosclerotic Disease. Amer. Heart Jour., in Press.

[4] Leriche, R.: The Surgery of Pain. Williams and Wilkins, Baltimore, 1939.

THE FIFTH BRAZILIAN CONGRESS OF ORTHOPEDICS AND TRAUMATOLOGY

Rio de Janeiro, June 30 to July 3, 1942

Editor's Note:—Unfortunately, we received the announcement of this meeting too late to include details of the program in our July issue. However, in the interest of Medicine in South America, we are privileged and pleased to print the following outline of the Congress.

The official reports to be discussed at the Congress are. Obstetric Paralysis and Internal Derangements of the Knee: the former having been contributed by Prof. Barros Lima and Dr. Orlando Pinto de Souza, and the latter by Prof. Domingos Define and Prof. Achiles de Araujo.

One of the sessions of the Congress will deal exclusively with War Traumatology. Various officials such as officers of the Army's Health Corps, Navy, Aeronautics, Fire Department and State Police having been invited thereto, as well as Civil elements. On this occasion Prof. Albee, Honorary Medical Colonel of the American Army and a well known traumatologist will lead a conference.